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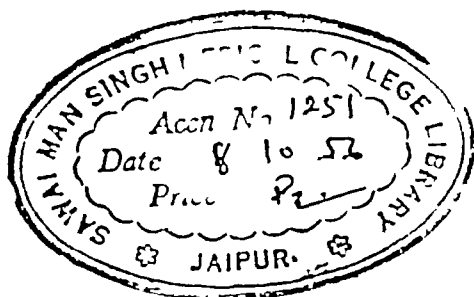
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FOUR YEARS CLINICAL EXPERIENCE WITH INTERNAL MAMMARY ARTERY IMPLANTATION IN THE TREATMENT OF HUMAN CORONARY ARTERY INSUFFICIENCY INCLUDING ADDITIONAL EXPERIMENTAL STUDIES

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SINCE our first publication¹ in 1946 we have repeatedly reported experimental evidence suggesting that it was possible to graft a third artery into the left ventricle and have it function in coronary artery insufficiency. Since our last report many points have been clarified and the technique of implant has been greatly improved. It is now four years since our first human case of coronary artery insufficiency was operated upon.

A short review of previously published experimental evidence will be given as well as recent information concerning blood flow studies made on the implanted internal mammary artery. The technique of implantation will be briefly outlined and our results on the treatment of twenty-nine human cases of coronary artery insufficiency given.

At this juncture let us consider two important questions: (1) Just how necessary is surgery in the treatment of coronary artery insufficiency? (2) What experimental evidence is needed before a procedure can justifiably be used on human beings?

A review of published statistics concerning the longevity and disability of patients with coronary artery insufficiency is most enlightening and most definitely disturbing. There is no question that every physician knows of patients who have lived many years with angina pectoris. However, an analysis of the average longevity in a series of cases throws a different light on this question.

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1. Bull. of the Society of Thoracic Surgery, Annual Meeting of The American Association for Thoracic Surgery, Montreal, Quebec, May 2-5, 1944.

In Table I the longevity of patients suffering from angina pectoris is shown for a group of 5027 cases reported in the recent literature. The average span of life in all cases is well under eight years and as Parker and associates¹¹ and Bruck and associates¹² who have reported the largest number of cases point out is probably five years. The figures quoted refer to angina pectoris without consideration of the presence or absence of coronary thrombosis with infarction.

TABLE I. LONGEVITY WITH ANGINA PECTORIS

AUTHOR	NUMBER OF PATIENTS WITH ANGINA PECTORIS	AVERAGE TO LONGEVITY (YEARS)
Horrobin and Nuzum 1918 ¹	200	0
MacKenzie 1927 ²	380	5.4 dead group
White 1926 ³	200	3.4 dead group
White and Plard 1921 ⁴	500 followed 2 to 7 years	7.9
	115 dead 52 alive	18.4
Uppinger and Fenn 1924 ⁵	141 fatal cases	1.7 average
Well and Smith 1924 ⁶	106 fatal cases	5.8 average
Farber et al. 1936 ⁷	140 Mayo Clinic study	5.0 entire group
Pringle, Crumpke and Dryden and Gage ⁸	5700 patients followed 5 to 22 years	15.6 dead end of first year 9% 1.4 each year thereafter Average 5 year survival 58.4% Entire group (normal pop. 86.9%) Average 10 years entire series 37.1% (normal pop. 70.4%)

In Table II is shown longevity after the first attack of coronary occlusion. The patients quoted by Conner and Holt⁹ and Levine and Rosenbaum¹⁰ may not have had the benefit of modern cardiologic methods of diagnosis. This, however, cannot be said of those quoted by Katz and associates¹ (see Table III) and Sigler.¹³ Although 81.5 per cent of Sigler's patients (males) and 83.1 per cent (males) were alive at the end of the second year only 45.4 per cent of the males and 49.8 per cent of the females were alive at the end of the fifth year. These figures have been taken from a rather large source of 1208 cases very carefully studied and cannot be minimized. Katz and associates¹ reporting 488 cases with infarction show a much more serious picture. They report that 25 per cent of their patients were dead at the end of two months, 50 per cent at the end of one year, and 75 per cent at the end of three years.

In Table III is shown the longevity in a series of cases that have had coronary artery occlusion with infarction

Now that we have discussed longevity in percentages, let us consider the ability of those who survive to work. There is no doubt that patients survive coronary artery occlusion with infarction, but what kind of lives do they lead? How many are capable of working and, if they are working how many of them are symptom free? According to Friedberg¹⁹ about 53 per cent return to work but of these only 32 per cent are able to work full time. Thus when

TABLE III PROGNOSIS WITH CORONARY ARTERY OCCLUSION WITH INFARCTION

AUTHOR	NUMBER OF PATIENTS WITH CORONARY THROMBOSIS	MORTALITY
Conner and Holt, 1930 ¹²	287	16.2 dead—first attack
Bedford, 1935 ¹⁴	31	6 months
Levine and Rosenbaum, 1941 ¹⁵	372 101 fatal cases	3.4 years
Master et al., 1936 ¹⁶	243 85% dead up to time of discharge	
Katz et al., 1949 ¹⁷	488 with infarction	25% dead, 2 months 50% dead end of 1 year 75% dead end of 3 years
Sigler, I. H., 1949 ¹⁸	1,208 423 died after one attack or more 785 alive after one or more attacks	Av longevity 3.8 yr (M) Av longevity 3.1 yr (F) Av longevity 4.9 yr (M) Av longevity 4.5 yr (F)
Pruck and Crumpruck ¹¹	1,302 cases—previous infarction	5 yr survival 45.9% 10 yr survival 26.0%
Dry and Gager ¹⁰	204 cases—subsequent infarction	5 yr survival 41.2% 10 yr survival 22.7%

we speak of approximately 50 per cent five-year survivals after coronary occlusion with infarction, one does not consider how many of these people who survived are useful citizens. In a general study of statistics one is surprised to find that only about 18 per cent of those who survive are able to return to a full and active life.

Now there is no doubt that every physician and every cardiologist knows of patients who have survived twenty years or longer following the onset of angina pectoris and who have worked and lived reasonably active lives. There are many such cases reported in the literature. These cases, however, are the exception rather than the rule as every general practitioner knows.

Thus it is clear that from the most optimistic figures less than 50 per cent of patients with one coronary artery occlusion will be alive at the end of five years and less than one third of those who survive the first attack are symptomatic and are capable of gainful occupation. It would seem from the figures presented that the disease progresses after the first coronary occlusion. Certainly this appears to be true between the first and fifth years after the initial attack (see Table II). Natural forces in the form of collaterals take over after the first coronary occlusion and maintain the myocardial muscle fibers in reasonable condition. Unfortunately the process of coronary artery sclerosis is progressive so that gradually more and more collaterals are shut

off and more and more muscle becomes fibrotic. Thus if a new source of blood can be brought to the myocardium before it is destroyed there is every reason to believe that further myocardial destruction can be stopped.

It must be remembered that the functional unit of the heart is the myocardial fiber. Once this is destroyed it cannot be replaced. When a great number of these fibers are destroyed the reserve of the heart is greatly diminished and eventually a condition is reached where so many myocardial fibers have been destroyed and replaced by fibrous tissue that the most ideal method of revascularization can do nothing to help such a heart. Therefore it is important to realize that the revascularization of the myocardium should be attempted as soon as possible to prevent progressive destruction and ultimate fatal loss of myocardial fibers. The onset of angina in severe form is in our opinion a definite indication for making an attempt at surgical revascularization of the myocardium. This will be discussed later under indications.

Experimental Criteria—It has often been said of experimentation that what is true of the animal does not necessarily apply to man and therefore that experimental evidence is of little value. This is partially true. However what is much more likely to be true is that if a procedure does not work in the heart of a dog or a pig it is not at all likely to be of value in the heart of a human being. Far too many new medical and surgical treatments have been tried out on human beings without having been adequately tested in the laboratory.

Many new drugs have been used in the treatment of angina pectoris which were at first hailed with enthusiasm only to be discarded in the light of cold facts. Likewise numerous surgical procedures have been devised each to gain a measure of popularity largely dependent upon the personality and energy of the surgeon who developed the procedure and each in turn like other form of treatment to be later abandoned. Practically all of these many therapeutic measures were founded on clinical and not upon laboratory data.

There are so many unknown factors in dealing with human coronary artery insufficiency that the value of any clinical method of treatment is most difficult to evaluate. Much time elapses and many lives are lost before the inadequacy of the treatment becomes apparent.

There is little question that before a method of surgical revascularization is attempted on human cases certain fundamental experimental facts must be ascertained concerning that particular method. In our opinion the minimal postulates that are necessary to prove the value of extracardiac circulation are five in number. These may be divided into (1) anatomical and (2) functional. The latter has four subdivisions—*a*, *b*, *c*, and *d*.

anatomical proof of the existence of anastomotic channels between the coronary arterioles and an extra cardiac source of blood in itself is not sufficient, it is necessary to prove its functional value

2 *Functional Value of an Extracardiac Blood Source* —

a *Survival following ligation of one of the major coronary vessels* Survival of animals following ligation of one or more coronary vessels without infarction gives important functional evidence as to the value of an extracardiac source of blood in replacing an obstructed coronary vessel. This is true, provided all occluded arteries are inspected afterward to make certain that (1) the occlusion is complete, and (2) that there are no branches proximal to the site of occlusion which are open. The latter is particularly true of ligation of the anterior descending branch of the left coronary. Frequently the septal branch arises directly from the left coronary artery. Ligation of the anterior descending branch in such animals cannot be considered complete. It has been our custom, when this occurs, to exclude such animals from both the control and treated series. In this way a much clearer picture is obtained with regard to survival following anterior descending branch ligation.

b *Interruption of the extracoronary circulation* When animals survive anterior descending branch ligation following a revascularization procedure, it is assumed that the majority survive because of the blood supplied to the myocardium from an extracardiac source. If this is true, then interruption of this extracardiac source of blood should result in immediate death, or survival with a large infarction.

c *Flow measurements of extracardiac blood source* It is important to be able to measure the amount of blood delivered to the myocardium by an extracardiac blood source. In addition it should be shown that the blood which flows into the heart is actually used by the functional unit of the heart—the myocardial fiber. To determine whether fresh arterial blood to the heart does not by pass the myocardium but is actually used by the heart muscle, oxygen consumption studies must be made.

d *Production and treatment of artificially produced coronary artery insufficiency* The production in animals of coronary artery insufficiency and its treatment by the introduction of an extracardiac source of blood is very important. This can be done by wrapping a piece of Dupont P-T 300 Cellophane around the origin of the anterior descending artery and slowly reducing, over several months, the exercise tolerance of the animal to a very low point. The successful introduction of an extracardiac source of blood into the ischemic myocardium should result in a return of the animal's tolerance to exercise as compared with the untreated and incapacitated animals.

Three different types of surgical procedure are now used in the treatment of human angina pectoris: (1) pericardial cardiopexy, (2) arterialization of the coronary sinus, and (3) internal mammary artery implant.

Before presenting the internal mammary artery method of revascularizing the ventricular myocardium which has been developed and tested in the experi

mental laboratories of McGill University during the past eight and one half years, let us examine the anatomy of the myocardium and the pathology of coronary artery disease

ARTERIALIZATION OF THE INTRAMURAL VESSELS OF THE LEFT VENTRICULAR MUSCLE BY MEANS OF INTERNAL MAMMARY ARTERY IMPLANTATION

Before proceeding to the details of this method for the revascularization of the ventricular muscle, a short review of the histological, anatomical, and pathological structure of the myocardium must be given. There are two factors now generally accepted upon which the procedure of internal mammary artery implantation is based—one is the anatomical pattern of the myocardial circulation, the other is the modern concept of the pathology of coronary artery sclerosis.

1 *Anatomical Structure of the Ventricular Myocardium*—It is well known that there are many differences between cardiac and skeletal muscles. The differences in which we are most interested concern the peculiarly rich network of blood vessels which supply the myocardial muscle fibers. Actually, there exists within the myocardium a veritable sponge-work of vessels. Their arrangement is graphically shown in Fig 1. As the smaller branches of the coronary arteries subdivide, they leave the surface of the heart and penetrate the myocardium at right angles. As they approach the endocardial surface they resume

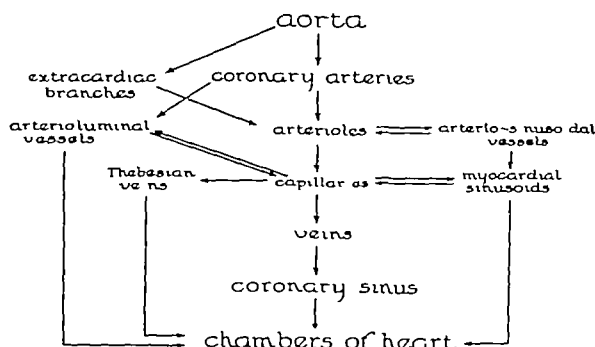


Fig 1—Relationship between the various components of the coronary circulation

then horizontal direction and arborize into a rich arteriolar bed from which the capillaries arise. The arterioles also communicate directly with sinusoidal arterial spaces which connect with the myocardial sinusoids. These latter vessels, with a lumen of from 50 to 250 μ in diameter, run a wandering course about the muscle fibers and anastomose with each other and the capillaries. Because of the structure of the myocardium, an implanted internal mammary artery with an open attached intercostal vessel fails to form a surrounding hematoma and remains patent for an indefinite period of time.

The second factor which indicates that internal mammary artery implant will be a successful method of treating coronary artery insufficiency is based on the pathologic character and distribution of coronary artery disease.

2 *Pathology of Coronary Artery Disease*—The generally accepted idea that coronary artery disease is widely spread throughout the coronary tree has not

been borne out by pathologic study. Arteriosclerosis of the coronary arteries is usually if not always confined to the epicardial part of their courses and never involves the penetrating myocardial branches to any significant degree. Thus, in coronary artery sclerosis there is a vast network of arterioles, etc., deep within the myocardium which are free of disease.^{21, 22} If arterial blood is delivered to this network, then it can reach all parts of the ventricular myocardium. An implanted internal mammary artery thus can bridge over the blocked superficial coronary vessels so that arterial blood can reach the intact deep arteriolar system of vessels which are still open and free of disease.

In the evolution of human coronary artery sclerosis a similar process occurs. It has been shown by Zoll²³ that in the normal human heart intercoronary anastomoses are rare. However, in the presence of arterial occlusion, intercoronary anastomoses develop distal to the point or points of occlusion. The development of such intercoronary anastomoses occurs frequently and probably prevents myocardial infarction when the occlusion develops slowly. Zoll²³ reports a study on a human heart with occlusion of the left anterior descending, left circumflex, and right coronary vessels, of such extent that the major portion of blood supplied to the heart came from a large separate conus artery which was connected with small branches below the points of occlusion in the descending coronary artery.

It is therefore, not surprising to find that an implanted internal mammary artery carrying a high pressure and placed beyond the point of coronary artery occlusion and among the deep myocardial vessels into a low pressure area is capable of vascularizing the left ventricular vessels. We feel that the term "collateral circulation" should be reserved for intercoronary anastomoses. Revascularization of the myocardium by collateral circulation can only occur provided one of the major coronary vessels is still patent. In the presence of major disease of the coronary vessels this can only occur through mechanical development of an extracoronary source of blood. This occurs so rarely under natural conditions that it is useless to await its development. The futility of waiting is borne out by the high mortality rate of coronary disease treated by medical measures only.

The use of the left internal mammary artery as an extracoronary source of blood supply was first attempted by us experimentally in 1945 and in 1950 we applied the procedure to our first human case of coronary artery insufficiency. The experimental data upon which the implant procedure is based have frequently been published but will be briefly reviewed here.

ANATOMICAL PROOF OF MAMMARY CORONARY ANASTOMOSIS

Procedure—The left internal mammary artery is freed from the chest wall from the fourth to the sixth intercostal space. The distal end is doubly ligated and transected between the ligatures. A tunnel is made in the anterior wall of the left ventricle and the freed portion of the artery is pulled into the tunnel and there fixed. The intercostal vessels which arise from the freed portion of the internal mammary artery are ligated except for the sixth which is transected just before the implant is pulled into the myocardial tunnel. The internal mammary artery is thus placed in a tunnel within the

ventricular myocardium, with an open, freely bleeding intercostal branch (Fig 2) In another technique, the artery is buried with its end open (Figs 6 and 7)

In previous publications^{1, 20} it was shown that an anastomosis developed between the implanted internal mammary artery and the left coronary circulation The presence of such mammary-coronary anastomoses in dogs was proved in the following manner

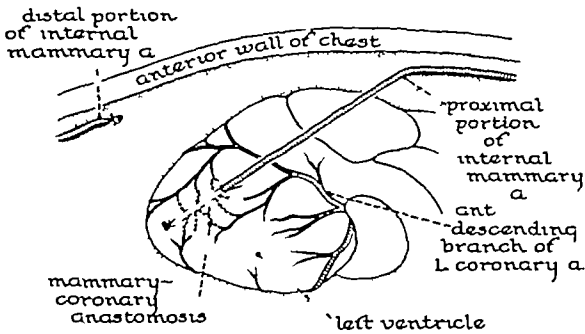


Fig 2—Internal mammary artery detached from its chest wall attachment and buried in a tunnel in the left ventricular myocardium Open stumps of intercostal not shown

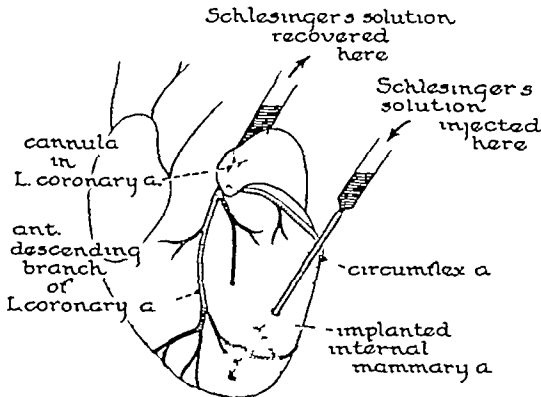


Fig 3—The method by which Schlesinger's Injection mass is introduced through a cannula placed in the internal mammary artery weeks to months after implantation In the presence of coronary mammary anastomosis the injection mass spreads throughout the left coronary arterial network and emerges from a second cannula placed in the mouth of the left coronary artery

Weeks or months after implantation the animals were sacrificed Two separate cannulas were used one was inserted into the internal mammary artery facing toward the heart the other was tied in the mouth of the left coronary artery The heart and attached internal mammary artery were removed from the thoracic cavity and Schlesinger's injection mass was introduced through the internal mammary artery This injection mass is known to pass freely through no vessel smaller than an arteriole 40 microns in diameter Where there was a large anastomosis the injection mass was seen to enter the

internal mammary artery, spread throughout the coronary arteries of the left ventricle and fill the cannula placed in the mouth of the left coronary artery (Fig 3). This would indicate that the anastomosis is with the arterial rather than the venous side of the coronary circulation. After injection, x-rays were taken to show the distribution of the injection mass throughout the coronary circulation.

Following x-ray studies sections were taken through the area of implantation and serial section studies were made. These serial sections showed branches leaving the implanted internal mammary artery and these branches could be followed out into the myocardium. In Fig 4 serial sections are shown which have been taken through the site of internal mammary artery implantation forty-eight days after implant. Note how the injection mass in the lumen of the implanted internal mammary artery can be followed out through a new branch into the myocardium.

Having proved the presence of mammary-coronary anastomosis by injection by radiographic studies and by serial section it was then decided to attempt visualization of the anastomosis by plastic casts.

From a few days to many months after implantation a plastic solution was injected into the proximal end of the internal mammary artery. At the same time similar plastic solutions of different colors were introduced into the left coronary artery and the coronary veins. The injection mass did not enter the capillary vessels. Acid and alkaline solutions were used for digestion and the resulting casts were studied under the dissecting microscope.

Two types of implants were studied: one in which the internal mammary artery was buried within the myocardium with its end open and the other with it closed but with an attached open bleeding intercostal branch. When the internal mammary artery is buried with its end open and bleeding into a myocardial tunnel an anastomosis occurs between it and the left coronary arterioles within five days. The entire left coronary arteriolar system can be filled with plastic substance through this implanted artery (Figs 5 and 6). Such anastomoses have been found to be present at least two and one half months after implantation (Fig 7).

When the internal mammary artery is buried with its end closed but with an open sixth intercostal artery, i.e. the standard implant described frequently during the past eight years, then the artery can be seen to branch at the end of twelve days (Fig 8).

These branches grow and at the end of three to four weeks are well-formed arterioles which join up with arterioles lying in the left ventricular myocardium (Fig 9). The anastomoses between the new branches from the internal mammary artery and the arterioles of the left coronary artery are, however, not usually fully developed under six weeks to two months.

In some of our injected specimens the casts show that the new branches join not only with the coronary arterioles but occasionally with the coronary veins (Fig 7).



Fig 4—Serial sections through site of internal mammary artery implant forty-eight days after implantation showing branching of transplant with infection in lumen

FUNCTIONAL VALUE OF MAMMARY-CORONARY ANASTOMOSIS

Histoanatomical proof of anastomotic channels between extracardiac arterial sources and the coronary circulation gives no indication of its functional value. This is true not only of coronary-mammary anastomosis but of all other procedures used for augmenting myocardial circulation. Functional tests give the best indication of the value of the new arterial blood supply. These may be divided into four groups:

A. Survival experiments following ligation of the anterior descending branch of the left coronary artery.

B. Removal of extracoronary circulation.

C. Experimental production and treatment of coronary artery insufficiency.

D. Blood flow measurements of the internal mammary artery after implantation.



Fig. 1.—Internal mammary artery (arrow) freed from subclavian to sixth intercostal artery and opened and implanted in a one-inch tunnel in left ventricular myocardium. Dog killed 14 days later. In right vinylite plastic inset 1 through internal mammary artery is seen the end of the artery of left ventricle.

A. Survival Experiments Following Ligation of the Anterior Descending Branch of the Left Coronary Artery—There is some divergence of opinion in the literature as to the effects of ligation of the anterior descending branch of the

left coronary artery. Some claim a low mortality for this procedure, and others claim a high mortality. The majority of investigators have found the mortality in dogs to range from 70 to 80 per cent. In our series of ten control dogs, the average mortality rate from anterior descending branch ligation was 80 per cent. It is our belief that the great variation in mortality reported in the literature following anterior descending branch ligation is dependent upon the site of ligation.

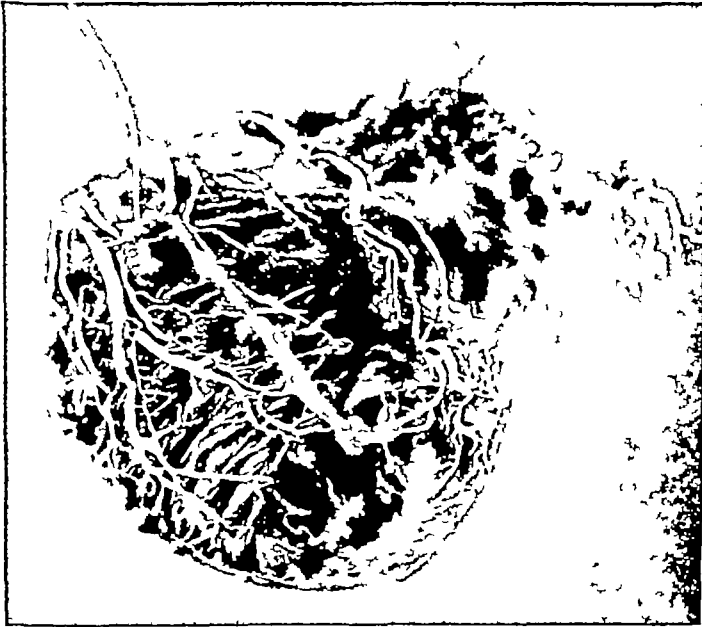


Fig. 6—Same cast as Fig. 5 except that the coronary vessels superficial to the implanted internal mammary artery were removed to show underlying internal mammary artery. Note how in five days vessels leave the implanted internal mammary artery to join the left coronary arteries.

Method of ligation. The anterior descending branch of the left coronary artery should be ligated at its origin. The position of the ligature must be carefully checked by examining the left coronary artery to make certain that the ligature has included all branches other than the circumflex. Frequently one or two septal branches are missed and occasionally branches arise from the main trunk of the left coronary artery.

An analysis of the results of anterior descending branch ligation is shown in Table IV. A total of 78 dogs had anterior descending branch ligation. There were 10 control animals and 30 other animals in which the coronary circulation was not augmented. In this group of animals there was an overall 80 per cent mortality with a 20 per cent survival with infarction. In 29 animals in which a large mammary coronary anastomosis had developed there were no deaths and no infarctions following anterior descending branch ligation.

TABLE IV EXPERIMENTAL DATA (VINEBERG AND ASSOCIATES)

	LIGATION ANTERIOR DESCENDING BRANCH OF THE LEFT CORONARY ARTERY			
	NO OF DOGS	DEATH	SURVIVAL WITH INFARCTION	SURVIVAL WITHOUT INFARCTION
Control	10	80%	20%	0
Internal mammary artery implant and/or other experiments but without augmentation of the coronary circulation	39	80%	3%	17%
Internal mammary artery implant with coronary anastomosis	29			100%

Internal mammary artery after implantation forms anastomoses with coronary circulation in from 30 to 75 per cent of animals operated upon

tion. Thus it would seem that coronary-mammary anastomosis not only increases coronary circulation but gives protection against death and infarction.

The experimental data shown in Table V indicate that coronary-mammary anastomosis when it occurs, is of value in protecting against death or infarction. However, because of the differences of opinion concerning the value of survival after descending branch ligation, a second group of functional experiments were performed.



Fig. 1. Internal mammary artery from chest wall from sixth intercostal artery to subclavian artery, distal end buried in 1/4 inch tunnel in myocardium of left ventricle. Distal end of origin and proximal end of descending branch anastomosed simultaneously. Chest opened 77 days after operation. Internal mammary artery isolated and injected with India ink. The ink was seen in the internal mammary artery anastomosis. Also mammary-venous anastomosis. Subclavian artery injected, plastic material into the internal mammary

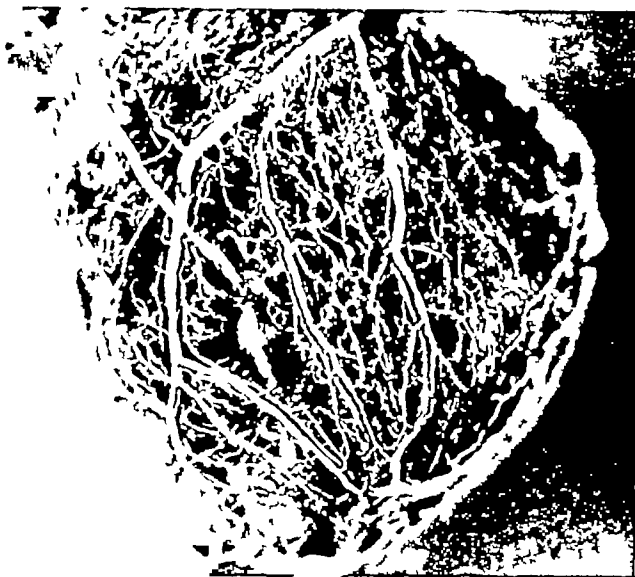


Fig 8—Internal mammary artery freed from chest wall between fourth and sixth intercostals and placed in 1 inch long tunnel in the ventricular myocardium with open stump of sixth intercostal. This animal died of pneumonia twelve days later. Cast made by injecting internal mammary artery with vinylite plastic and cut away to show buried internal mammary artery with stump of sixth intercostal (arrow). Note tufting and branching from open stump of sixth intercostal attached to internal mammary artery.

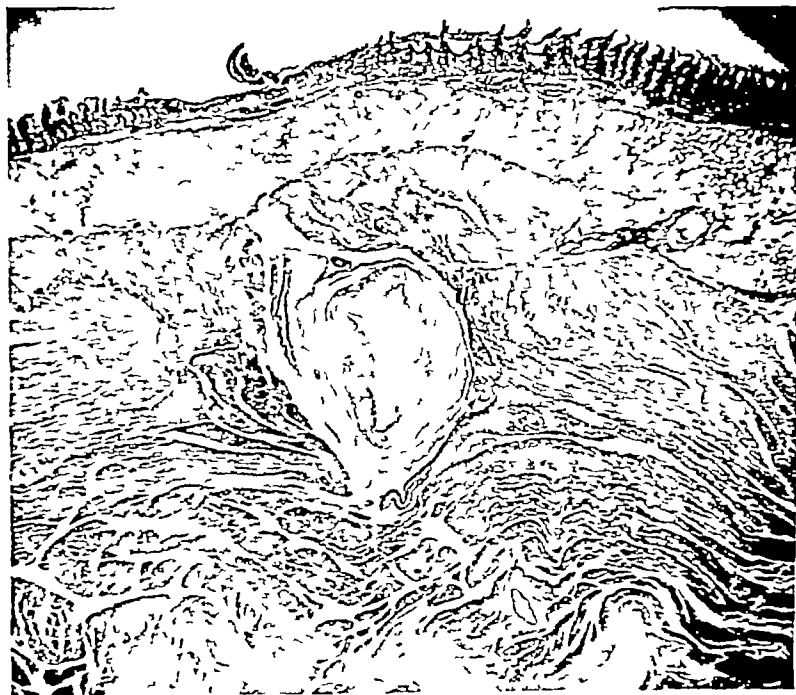


Fig 9—Section through human heart at site of internal mammary artery implant 62 hours later. Note how the vessel lies open between the myocardial fibers and the absence of surrounding hematoma. This is remarkable because the artery was pulled into position with an attached open bleeding intercostal vessel. The absence of clot in the lumen of the vessel is likewise to be noted.

B Removal of Extracoronary Circulation—Animals which survived anterior descending branch ligation of the left coronary artery were subjected, four to five weeks later, to complete and sudden occlusion of the implanted internal mammary artery. If the internal mammary artery was maintaining the circulation of the left ventricle, then following ligation either death or infarction should result. This is exactly what happened. In three of the animals with an internal mammary artery implant which had survived anterior descending branch ligation the internal mammary artery was ligated. One animal died immediately. Another animal died within twenty-four hours and displayed an edematous, cyanotic area of the anterior wall from a large infarct in the same location. Another animal survived, but examination of the sacrificed specimens revealed that multiple intercoronary anastomoses were present.

In all these animals there was a demonstrable mammary-coronary anastomosis. There can be little doubt that in those animals which died following internal mammary artery ligation the circulation of the anterior portion of the left ventricle was being maintained by the internal mammary artery implant.

The evidence just presented suggested that the internal mammary artery after implantation into the left ventricular wall is capable of maintaining the left ventricular circulation. These experiments, however, were carried out on the hearts of normal animals. Therefore, it was necessary to attempt to artificially produce coronary artery insufficiency and to treat it by internal mammary artery implantation. Thus we set out to experimentally produce and treat coronary artery insufficiency and so establish a third functional test of the value of an internal mammary-coronary anastomosis.

C Experimental Production and Treatment of Coronary Artery Insufficiency—In this series of experiments coronary artery insufficiency was produced in dogs and treated by internal mammary artery implant. The first problem was to find a method of producing coronary artery occlusion which would simulate that which occurs in human coronary sclerosis. The second problem concerned the evaluation of coronary insufficiency when it was produced and its response to treatment.

Method of producing coronary artery insufficiency—The anterior descending branch of the left coronary artery was exposed at its origin. Beneath the left atrioventricular sulcus to the left of the pulmonary trunk the epicardium overlying the vessel was incised and the bifurcation of the main coronary artery exposed. A doubly folded strip of Cellophane (Dupont-P-T 700) sterilized for twenty-four hours in a saturated solution of mercuric oxyamide was twice wrapped completely but loosely around the origin of the anterior descending branch of the left coronary artery and fixed in position. Thus a segment of artery from 4 to 8 mm long was completely surrounded with Cellophane.

The Cellophane initiated a fibroplastic reaction with collagen deposition about the artery. As subsequent contraction of the new fibrous tissue

occurred, the lumen of the anterior descending branch of the left coronary artery was slowly narrowed. Gradually, that portion of the myocardium nourished by this vessel became ischemic.

At the end of the experiment when the animals were sacrificed, the anterior descending branch of the left coronary artery was probed to determine the extent of the narrowing. Sections were made through the artery at the site of the Cellophane wrap, and careful histologic studies made of the degree of coronary occlusion.

Method of evaluation of experimentally produced coronary insufficiency and its treatment. Exercise tolerance test. In human coronary artery insufficiency there are two outstanding symptoms, namely, substernal pain, and intolerance to exercise. In the dog, it is most difficult to detect the presence of pain. It is, however, not difficult to determine accurately the animal's tolerance or intolerance to exercise.

Clinical and pathologic experience have associated progressive exercise intolerance with progressive myocardial ischemia. Thus, in the experimental animal, progressive diminution of exercise tolerance has been accepted as evidence of myocardial ischemia. In this series of experiments, myocardial ischemia was produced by a Cellophane wrap placed around the origin of the anterior descending branch of the left coronary artery. Exercise tolerance studies were carried out before and after Cellophane wrap. Later, after internal mammary artery implant, they were repeated in order to establish whether improvement had occurred.

A treadmill was used to exercise the animals. This was made originally for man and was thus very satisfactory for exercising large animals. The exercise tolerance of each animal was established prior to wrapping the anterior descending branch with sclerosing type Cellophane. Two groups of animals were studied. The first group was composed of those animals in which coronary artery insufficiency was produced by Cellophane occlusion of the anterior descending branch, and treated by internal mammary artery implant. In this series the average running time was reduced from 9-12 minutes to 2-5-4 minutes, at 8½ miles per hour three months after Cellophane wrap. These animals were given an internal mammary artery implant and three to five months later, the running time had returned to 6 to 8 minutes respectively, whereas the control animals had an average running time of 16 minutes at 8½ miles per hour. In those animals with an implant in which the running time had returned there was a wide open coronary mammary anastomosis.

In the second group of animals an internal mammary artery implant was performed at the same time as the Cellophane wrap around the anterior descending branch. Where there was a widely patent mammary coronary anastomosis there was no appreciable reduction in exercise tolerance.

D. Blood Flow Measurements of the Internal Mammary Artery After Implantation.—Measurement of the amount of blood delivered to the heart by an implanted internal mammary artery, five, six, and seven months after im-

plantation have been made by means of a graduated biochemical burette a stop watch and a pressure tank (Fig 10). The left subclavian and internal mammary arteries are isolated the latter down to its point of entry into the heart and a cannula placed in the subclavian artery. The outlet of the burette is connected with the cannula in the subclavian artery and the top of the burette to a pressure tank. The time taken for 10 cc of blood to flow

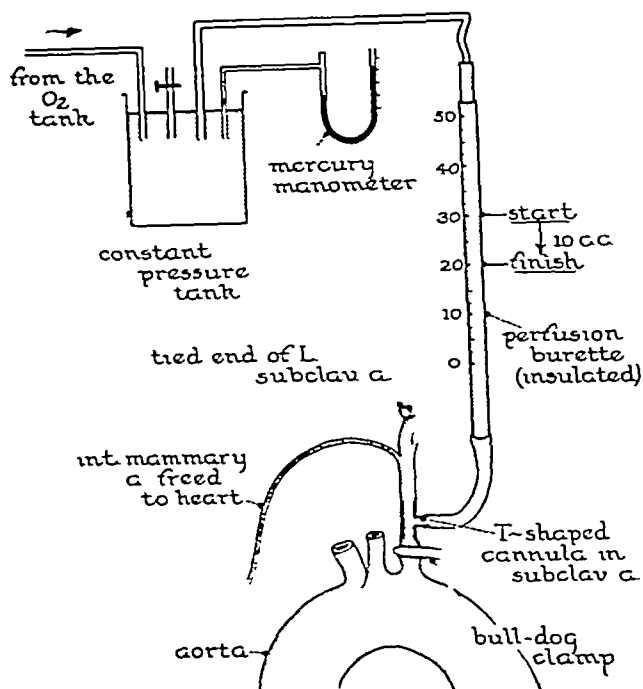


FIG. 10—A diagrammatic representation of the perfusion apparatus for the measurement of blood flow through the implanted internal mammary artery. The rate of blood flow per minute is determined by clocking with a stop watch the time taken for 10 cc to flow through the internal mammary artery. Flow rates at pressures equal to above and below the carotid blood pressure were studied.

into the heart through the internal mammary artery was measured by a stop watch is expressed in cubic centimeters of flow per minute. The flow rate has been studied at perfusion pressures above, below, and equal to the carotid blood pressure.

The internal mammary artery is capable of delivering 85 cc of blood per minute (Table V) with both its ends and the sixth intercostal artery open. When the distal end of the internal mammary artery is tied and only the sixth intercostal is open—as it is when it is pulled into the myocardium—the perfusion rate is 45 cc per minute.

In four dogs studied five to seven months after implantation and four to six months after ligation of the anterior descending branch the flow rate varied from 5 cc to 21 cc when the perfusion pressure was equal to the carotid blood pressure and from 14 cc to 51 cc when the perfusion pressure

was higher than the blood pressure. The blood flow through an implanted internal mammary artery into the heart five months after implantation is well shown in Table VI. The rate of 21 cc per minute when the blood and perfusion pressures are equal is indeed a functional amount of blood to be delivered to a heart via an implant. By increasing the perfusion pressure in relation to the blood pressure, the amount of blood sent into the heart via the internal mammary artery implant reached 51 cc per minute.

TABLE V BLOOD FLOW STUDIES (INTERNAL MAMMARY ARTERY IMPLANTATION FOLLOWED BY LIGATION OF ANTERIOR DESCENDING BRANCH SIX WEEKS LATER)

ANIMAL NO	CONTROL	TIME AFTER IMPLANT	CAPOTID B/P (MM HG)	SUBCLAVIAN ARTERY PERFUSION PRESSURE (MM HG)	FLOW (CC PER/ MIN)	RETROGRADE FLOW (CC PER/ MIN)	ANASTOMOSIS
1	Internal mammary artery open end + open 6th intercostal		60	60	85		
	6th intercostal only		60	60	45		
12S		7 months	0	120	11	Heart started to beat	Large
521		6 months	100	131	3		
			100	231	14		
			50	131	5	Slow retrograde	Small branches
			65	0			
15		5 months	100	137	7		
			75	77	3		Large
			65	0		13	
33W		5 months	102	123	29		
			96	93	21		Large
			98	23		1	

Oxygen Saturation Studies—After making the measurements just described the animal was bled to death, the heart stopped, and a cannula was tied in the coronary sinus. Oxygen saturation measurements were made of the blood perfusing the internal mammary artery and recovered from the coronary sinus (Table VII). Table VII shows these figures and also demonstrates how the oxygen saturation dropped from 98 to 40 per cent after traversing the vessels of the heart. At this point the heart began to beat rhythmically again. It would seem that blood which goes through an implanted internal mammary artery enters the myocardial arteriolar system via mammary coronary anastomoses and then enters the capillary or myocardial sinusoidal system where it yields its oxygen to the myocardial fibers and is returned in part via the venous and coronary sinus systems. In the experiment shown in Table VIII, sufficient oxygen was supplied to the myocardium through a 5 month old internal mammary artery implant to an arrested heart to make it beat again.

The character of the mammary-coronary anastomosis five months after implantation and three and one-half months after ligation of the anterior

descending branch is shown in Fig 11. Through these branches from 21 to 51 cc of blood per minute was delivered to the heart (Table VII). Sufficient oxygen was supplied to the myocardial fibers to start the heart beating again after its arrest.

TABLE VI. BLOOD FLOW THROUGH IMPLANTED INTERNAL MAMMARY ARTERY FIVE MONTHS AFTER IMPLANTATION AND FOUR MONTHS AFTER LIGATION OF ANTERIOR DESCENDING BRANCH

CAROTID ARTERY PRESSURE (MM. HG.)	INTERNAL MAMMARY ARTERY PRESSURE AFTER CORRECTION FOR HEIGHT OF BLOOD COLUMN IN PERFUSION BUTETTE	FLOW THROUGH IMPLANTED INTERNAL MAMMARY ARTERY INTO HEART (CC/MIN.)
102	123	29
96	93	21
92	63	10
90	33	3
88	23	minus 1 (reverse flow)
<i>Elevation Perfusion Pressure Above Carotid Blood Pressure</i>		
86	123	32
86	153	43
82	183	51

(Dog 33W)

TABLE VII. MYOCARDIAL CONSUMPTION OF OXYGEN FROM BLOOD SUPPLIED THROUGH THE IMPLANTED INTERNAL MAMMARY ARTERY FIVE MONTHS AFTER IMPLANTATION

CAROTID PRESSURE (MM. HG.)	INTERNAL MAMMARY ARTERY PERFUSION PRESSURE (MM. HG.)	BLOOD FLOW THROUGH INT. MAMMARY ARTERY IMPLANT (CC/MIN.)	CORONARY SINUS FLOW (CC/MIN.)	OXYGEN SATURATION		REMARKS
				INT. MAMM. BLOOD (PER CENT SATURATION)	CORONARY SINUS BLOOD (PER CENT SATURATION)	
0	123	54	30	98	60	
0	122			98	55	
0	123			98	55	
0	123			98	42	
0	123			98	40	Heart started to beat

DURATION AND CHARACTER OF CORONARY-MAMMARY ANASTOMOSIS

Glenn and his associates²⁷ have confirmed the development of an anastomosis between an implanted internal mammary artery and the coronary vessels. However they stated that the new vessels tend to disappear at the end of eight weeks. They further suggested that the new vessels were small and resembled granulation tissue. In our series the average interval from implant to sacrifice was eleven weeks (Table VIII). One animal was kept for fifty-eight weeks after implantation. The anastomosis at the end of that time not only persisted but were large enough to protect against death and infarction following ligation of the anterior descending branch.

Not only has it been suggested that a coronary-mammary anastomosis tends to disappear at the end of eight weeks but it has also been suggested that the anastomosis is capillary in nature. Serial sections of seven implants have been examined. Special preparations of these serial sections have been

made to show elastic and muscle tissue. The character of the new branches formed by the internal mammary artery implant has thus been studied. They have been found to be arterial in nature, containing both muscle and elastic tissue in their walls. These new arterial branches have been followed by serial section far out into the myocardium until they disappear.²



Fig. 11—This is a cast of the heart of Dog 33W, flow measurement studies of which are shown in Table VI and oxygen consumption studies in Table VII. This animal had an internal mammary artery implant five months before the cast and flow studies were made and had survived anterior descending branch ligation three and one half months previously.

FREQUENCY OF MAMMARY-CORONARY ANASTOMOSES

We have heard the odd whisper that "the internal mammary artery implant is excellent when it works, but that most of the implanted arteries are blocked." In order to satisfy ourselves as well as others, we have reviewed

TABLE VIII DURATION OF ANASTOMOSIS

TYPE OF ANASTOMOSIS	NO. OF ANIMALS	PRESENCE OF ANASTOMOSIS AFTER IM PLANT (WEEKS)	VALUE OF ANASTOMOSIS AFTER LIGATION OF ANTERIOR DESCENDING BRANCH OF THE LEFT CORONARY ARTERY
<i>Microscopic Anastomosis</i>	1	5	
	1	6	
	1	7	
Anastomosis small due to thickened or partially thrombosed internal mammary artery	2	8	Protection against death or infarction afforded in three animals only
	2	11	
	2	12	
	2	13	
	1	14	
	12		
<i>Macroscopic Anastomosis</i>	1	6	
	2	7	
	5	9	No deaths or infarctions occurred
Anastomosis large	5	11	
	4	12	
	2	14	
	1	17	
Internal mammary artery patent	1	18	In coronary artery sclerosis caused by Cellophane irritation marked improvement in exercise tolerance occurred
	2	19	
	1	21	
	1	31	
	1	41	
	1	46	
	1	58	
	29		

TABLE IX FREQUENCY OF MAMMARY CORONARY ANASTOMOSIS STANDARD VINEBERG IMPLANT

	NO. DOES	ANTERIOR DESCENDING BRANCH LIGATION	SURVIVED	WITHOUT INFARCT	ARTERY OPEN	ANASTOMOSIS
NO MAMMARY HEART	9 (Vineberg)	Not done	2	1	4	4
	6 (Niloff)	6	2	1	2	1
	11 (Miller)	11	3	1	1	8
	13 (Buller)	13	5	5	12	5
Total	39	30	10 (70%)	12	25 (65%)	18 (46%)
	11	Partial ligation and desc. branch (23 gauge needle)	8 (72.7%)	5 (45.5%)	7 (63.6%)	6 (54.5%)
ISCHEMIA MAMMARY	50	Cellophane wrap ant. desc. branch followed by implant 3 to 4 months later	Exercise tolerance markedly reduced before I.M. implant. It turned nearly to normal in 2. Further reduced in one (abscissae). Not tested in 2 (compensated).		4 (80%)	4 (80%)
	50	Cellophane wrap ant. desc. branch				Condition of artery not recorded in 2
	10	Implant in coronary artery	2—Exercise tolerance reduced for 8-21 min		2 (100%)	2 (40%) small
	10		2—Exercise tolerance maintained at preop levels		2 (100%)	2 (60%) large
Total	149				16 (76%)	15 (71%)

our experimental data concerning the frequency of mammary coronary anastomoses in a series of sixty animals in which the standard internal mammary artery implant procedure was performed (Table IX)

Assuming that the proper technique is employed, the frequency of coronary-mammary anastomosis appears to be related to the heart's demand for fresh arterial blood. Thus, in thirty-nine animals receiving an internal mammary artery implant in normal hearts, there were eighteen (46 per cent) mammary-coronary anastomoses, and only 30 per cent of these protected against death from anterior descending branch ligation (according to our standards). Twenty-six (67 per cent) of the implanted arteries remained open many weeks to months later. When the hearts were made somewhat ischemic by partial ligation of the anterior descending branch, the anastomotic rate was 54 per cent and the survival rate from anterior descending branch ligation was 72 per cent. Myocardial ischemia produced by wrapping Cellophane around the origin of the anterior descending branch simultaneously, or followed by internal mammary artery implant, resulted in an 80 to 100 per cent anastomotic rate. The average anastomotic rate in normal hearts was 46 per cent with 67 per cent open vessels. In ischemic hearts the anastomotic rate averaged 71 per cent, with 86 per cent open vessels. This could be explained as follows:

The chance of an internal mammary artery implant branching will depend theoretically on its being placed in an area where the pressure is somewhat lower than within itself. The greater the differential pressure between the pressure within the implant and the pressure in the heart the greater is the likelihood of the artery branching. Thus when it is placed in normal heart muscle the pressure differentials are not as great as when it is placed in an ischemic heart muscle with a low pressure area. On this basis, the higher frequency of mammary-coronary anastomoses in ischemic hearts can be explained. It is likewise logical to deduct that patients suffering from coronary insufficiency should have about the same anastomotic rate as that seen in ischemic animal hearts.

TREATMENT OF HUMAN CORONARY ARTERY INSUFFICIENCY BY INTERNAL MAMMARY ARTERY IMPLANTATION

On the basis of the foregoing anatomical, functional and pathological facts the internal mammary artery implant procedure has been used in the treatment of human cases of coronary artery insufficiency.

Selection of Patients.—The selection of patients for a new procedure is always difficult. We dislike classifications but in a general way we have found it convenient to divide our cases of coronary artery insufficiency into four groups:

Group 1 Mild or intermittent angina. In this group are those patients who experience an occasional attack of anginal pain on exercise, or during

in emotional disturbance. They are able to work and are capable of living reasonably normal lives. For this type of patient operation is not advisable—it is better to stop playing golf!

Group 2 Progressive coronary artery insufficiency without disability Evidence of progressive coronary artery insufficiency is an important indication for surgery. Patients in this group may still be working, but there is evidence of progression of their disease. This may be shown in the electrocardiogram, or by an increase in the frequency of anginal attacks and, perhaps, progressive dyspnea.

Group 3 Partially or totally disabled, but without angina at rest In this group are hundreds of thousands of people, men and women, who are unable to walk more than 50 to 100 feet, or to do 22 steps on the Master two-step test without anginal pain. Many suffer pain at rest, but in association with emotion, straining at stool, or a heavy meal. Some have night pain which awakens them. However, when sitting quietly in a chair, without stimuli, they are free of pain. Such cases have been accepted for surgery; in fact, surgery should be encouraged as it has been shown that less than 50 per cent of such patients are alive at the end of five years. Another coronary artery occlusion may so lower their myocardial reserve as to place them in the hopeless inoperable group of patients with status anginosus.

Group 4 Status anginosus In this group are patients who experience angina at rest. These patients chew nitroglycerin tablets day and night. One of our patients in this group consumed 180 nitroglycerin tablets in two weeks. This type of patient should not be submitted to cardiac surgery. There is insufficient cardiac muscle left to revascularize. Generally, these patients are living on a pinpoint opening in the one artery which is still open. Such a small opening usually cannot supply sufficient blood to the myocardium to keep the patient alive during and after a major thoracic procedure. In addition, such patients generally have very little myocardial muscle left to revascularize.

Preoperative Investigation—With each patient we have attempted to exclude all other possible causes for his symptoms. This has been done in spite of a well established diagnosis of coronary artery insufficiency. Thus a search is made for intervertebral discs, infradiaphragmatic and supradiaphragmatic lesions and in all cases a psychiatric screening is done. A careful examination of serial electrocardiograms must be made for evidence of recent activity as well as repeated sedimentation rates. In a few cases we have postponed operation for from four to six months on evidence of activity.

Contraindications—(1) Evidence of left ventricular failure. The history of left ventricular failure during an attack of myocardial infarction with recovery is not a contraindication. However, the presence of left ventricular failure most definitely is a contraindication. (2) Enlargement of the left ventricle unless it is due to hypertrophy associated with moderate hypertension. (3) Advanced cases of essential hypertension. (4) Evidence of recent myocardial infarction or of active disease. (5) Status angina.

PREOPERATIVE, OPERATIVE, AND POSTOPERATIVE CARE

Having determined that the patient has no contraindications to surgery, quinidine sulfate, 3 grains by mouth, is given twice on the preoperative day and once on the day of operation. An enema is given early in the afternoon of the preoperative day as we have found that many patients are disturbed by it and have their sleep interrupted. Sodium luminal is administered, 3 grains, at 10 P.M., 11 P.M., and again at 6 A.M. Luminal sodium does not depress the blood pressure as other barbiturates do. At 7 A.M. on the day of operation, morphine or Demerol and 1/100 gram of atropine are given. It is very important that anesthesia be administered as soon as the patient arrives in the operating room. These patients generally are apprehensive and may develop a fresh coronary occlusion while awaiting operation. The surgeon must check to make certain that the patient is not suffering substernal pain just before the anesthetic is started. We failed to take notice of this warning signal in our first sixth and eighteenth patients. The first two patients died within sixty-two hours after operation from fresh coronary occlusions, the third developed pulmonary edema after the chest was opened and died eleven hours later.

Upon arrival in the operating room, the patient is given oxygen to breathe and at the same time a 2 per cent procaine hydrochloride drip is started. Induction is achieved with cyclopropane and anesthesia maintained with ether and oxygen. Once the patient is asleep a cut-down is started in the ankle; this is essential in case of a drop in blood pressure.

BLOOD PRESSURE MAINTENANCE

The maintenance of blood pressure is, we feel, extremely important. Neo-Synephrine drip is used when necessary and the blood pressure is maintained at a constant level throughout the operation. The desired blood pressure level is decided upon before operation and is based on the average blood pressure levels recorded four times daily during the ten days preceding operation. We have attempted to maintain blood pressure levels at not less than 20 mm. Hg below average preoperative levels. The coronary blood flow is dependent upon the diastolic blood pressure. Patients with coronary artery insufficiency are not like those patients with other types of heart disease in whom the coronary arterial system is comparatively healthy. Lowering of the diastolic pressure decreases the blood flow through the coronary vessels and lessens the amount of blood supplied to the myocardium. When this occurs in the presence of narrowed calcified coronary arteries we believe that coronary thrombosis is favored. In addition the myocardium which is already suffering from ischemia receives insufficient blood for the maintenance of life, and the development of left ventricular failure and pulmonary edema are therefore favored.

FAILURE TO MAINTAIN BLOOD PRESSURE

The blood pressure in some of our cases has fallen with induction and in others after the patient is positioned on his right side. If the blood pressure

cannot be returned to proper levels with a slow Neo-Synephrine drip of 5 cc in 500 cc of 5 per cent glucose and water then the operation is not begun. This has happened in two of our patients neither of whom was operated upon.

OPERATIVE TECHNIQUE

A paper describing our operative technique in greater detail than is possible in this article is now being prepared.

The patient is placed in the right lateral position, and the fifth or sixth rib is removed depending on the location of the apex. Procaine, 6 cc of 1 per cent in normal saline is injected into the pericardium. The internal mammary artery is freed from the fourth to the sixth intercostal spaces inclusively. It is doubly ligated at its distal end and cut between ligatures. The mesial and diaphragmatic fat pads are cut away from the fibrous pericardium and left attached mesially and inferiorly respectively. The fibrous pericardium is removed over the entire anterior surface of the heart and part of the inferior surface. The heart is inspected for areas of infarction and fibrosis and the left coronary anterior descending branch and circumflex arteries are palpated for disease which can be felt very readily.

A suture of No. 10 cotton is placed in the heart distal to the area selected for tunneling the myocardium. Proximal to this suture a small incision is made in the epicardium. A tunnel of $\frac{3}{4}$ to 1 inch is made by blunt dissection using a curved mosquito artery forceps. The cotton attached to the tied end of the proximal end of the severed internal mammary artery is picked up with the mosquito forceps and pulled through the tunnel. The sixth intercostal artery attached to the internal mammary artery is then cut and allowed to bleed freely. The internal mammary artery and bleeding sixth intercostal arteries are pulled into the myocardial tunnel. The end of the internal mammary artery is pulled out of the distal end of the tunnel where it is secured by the first suture placed in the heart.

Pericardial Fat Graft as Supplement to Internal Mammary Artery Implant—During the past five years we have used various techniques of implantation hoping to increase the rate of mammary coronary anastomosis. One of these first reported by Vinberg and Nisloff 1950,²⁰ was the pericardial fat wrap. At that time we thought the internal mammary artery tended to angulate less when surrounded by the pericardial fat. Subsequent experiments were not confirmatory. Bigelow* revived this idea and pointed out the necessity of preserving the blood supply of the pericardial fat grafts. Since October 1953 we have pulled the internal mammary artery through the mesial pericardial fat pad before implantation. In addition we have used the pericardial fat pads as a supplement to the internal mammary artery in revascularizing the ventricular myocardium (Fig. 12) whenever possible. After the artery has been pulled through the mesial pericardial fat pad and implanted, areas of epicardium are removed on the anterolateral surface of the heart. The mesial fat pad is then sutured directly to the exposed myo-

cardial muscle. The inferior diaphragmatic fat pad is likewise sutured to a similar area on the posterior surface of the heart which has been denuded of its epicardium*.

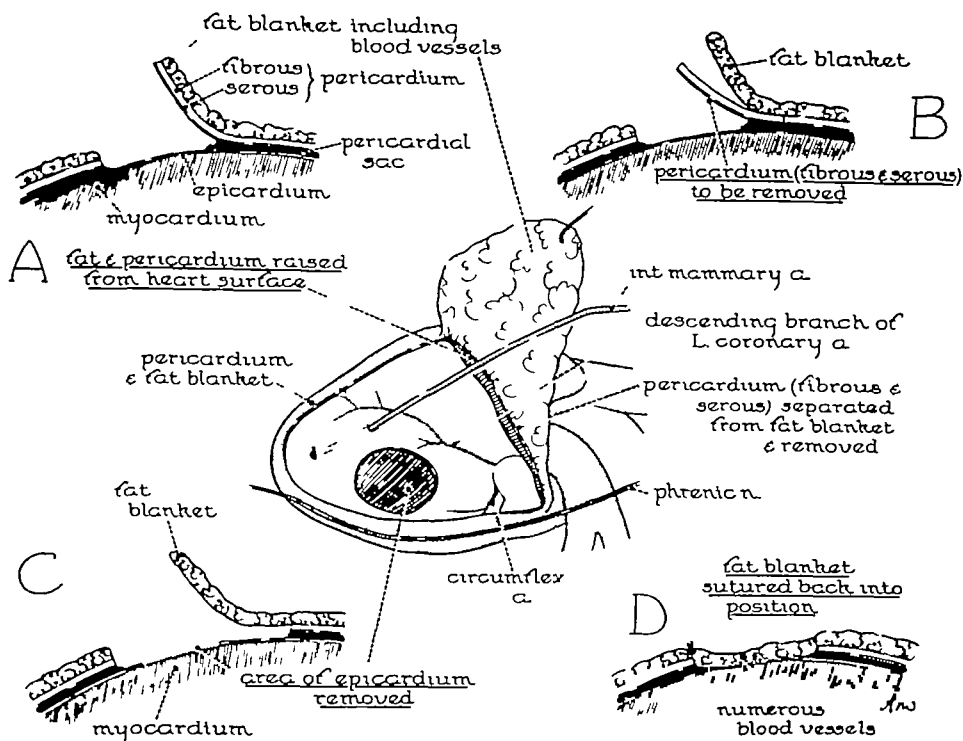


FIG. 1.—1 Pericardium composed of fat layer on top of a fibrous layer which is lined by parietal endothelium. Epicardium a thin layer covering the surface of the heart which can be removed, exposing heart muscles. B fat layer separated from fibrous pericardial layer. C removal of fibrous pericardial layer and epicardium. D placement of vascular fat blanket on denuded ventricular surface. Center showing relationship of fat blanket to implanted internal mammary artery.

POSTOPERATIVE CARE

The two most important features of postoperative care are the maintenance of blood pressure at a constant level, and the maintenance of fluid and electrolyte balance. Neo-Synephrine continuous drip is used at 20 to 30 drops per minute depending on the weight of the patient, starting with 5 cc Neo-Synephrine in 500 cc of 5 per cent glucose and water. Sometimes this may have to be increased as much as 25 cc per 500 cc of 5 per cent glucose and water.

Patients are not moved for forty-eight hours. A movement of even 15 degrees can cause a marked drop in blood pressure. After forty-eight hours patients can be moved from the back to the right side, never to the left side. The amount of blood pressure instability seems to depend on the extent of

*For details of our first patient see preliminary report of the first human case operated upon using pericardial fat pads as a supplement to internal mammary artery implant. Canad. Med. Ass. J. 70: 767-78, 1954.

myocardial damage present before operation, as indicated by the exercise tolerance. Patients with low exercise tolerance invariably need blood pressure support for two, and sometimes five and six days. Others, with exercise tolerance of 20 to 29 steps, seem to require very little vasopressor help.

Neo Sympheprine aerosol is used every two hours as soon as the patient awakens from the anesthetic. Wangenstein suction is started early as some patients develop abdominal distention. This is discontinued as soon as there are abdominal cramps and passage of gas. Nasal oxygen is used routinely for twenty-four hours and longer if necessary. Quinidine sulfate is given by hypodermic, 3 grains twice daily. Usually, after the third day, Luminal sodium is substituted for Demerol. No hot or cold fluids are given, and the bedpan is never permitted. Mineral oil is started on the third day, as is Dicumarol. Usually patients are permitted out of bed on the sixteenth to the twenty-first day, and can usually leave for home around the fourth post-operative week.

RESULTS

This report deals with our experience with 29 patients who have undergone the internal mammary artery implant operation. This operation was completed in 28 patients. These cases have been divided into two groups—the first group of 10 patients followed for 1 to 4 years, and the second group of 19 patients followed from 2 to 11 months.

In the first group of 10 patients (Table X) all were disabled from 7 to 36 months, the average period of disability being 21 months. Half of these patients had had anterior and posterior infarctions, and only two patients showed no evidence of infarction. Exercise tolerance ranged from 50 to 200 feet. In this group followed one to four years after operation (Table XI) 3 patients died within 62 hours after operation, all 3 were cases of status angina. Seven survived the operation. Five or 71 per cent, have returned to work and are working from 8 to 16 hours daily. One is working 16 hours daily and has slight pain on lifting or fast walking three years postoperatively. One worked for two years and then died pushing his car out of the snow, death was due to rupture of an old myocardial scar. Two still have angina postoperatively. One lived three years and died of aplastic anemia, the artery having pulled out of the heart. One is still alive and is showing improvement at the end of one year.

In the second group of 19 cases (Table XII) followed 1 to 10 months, there were only 3 patients in whom there was no apparent infarction. Two patients had anterior and posterior infarctions. Six were working at sedentary jobs or with the aid of nitroglycerin. Thirteen were totally disabled from 2 to 21 months. There were 3 cases of angina status. Of these 19 patients, the operation was not completed in one, and there was one death (Table XIII). Seventeen patients underwent internal mammary implant and 10 of these had the supplementary procedure of pericardial fat pad graft.

TABLE X FIRST TEN HUMAN CASES OF CORONARY ARTERY DISEASE FOLLOWED ONE TO FOUR YEARS

	SEX	AGE	OCCUPATION	TIME DISABLED (MONTHS)	EXERCISE TOLERANCE	EATING	NIGHT	DYSPNEA	FATIGUE	PREVIOUS INFARCTI ON
	<i>No Anginal Pain at Rest</i>									
	M	54	Oil field worker	7	150 ft	Yes	No	Yes	Yes	None
	M	49	Executive	24	100 ft	Yes	No	Yes	No	Anterior posterior
	M	55	Machinist welder	24	150 ft	Yes	No	No	No	Large anterior
	M	55	Grocer	10	50 ft	Yes	No	Yes	Yes	None
	M	57	Electrician	10	50 ft	No	No	No	No	Large anterior
	M	52	Farmer	28	200 ft	No	No	Yes	Marked	Posterior
	M	39	Physician	24	50 ft	Yes	Yes	Yes	Marked	Anterior posterior
	<i>Status Angina</i>									
	M	53	Tailor	36	zero	Yes	Yes	No	No	Anterior posterior
	M	57	Civil servant	36	100 ft	Yes	Yes	Yes	No	Anterior posterior
	M	46	Stevedore	12	150 ft	Yes	Yes	Yes	No	Anterior posterior

TABLE XI FIRST TEN HUMAN CASES OF CORONARY ARTERY DISEASE FOLLOWED ONE TO FOUR YEARS

NAME	TIME SINCE OPERATION (MONTHS)	OCCUPATION	ANGINAL PAIN	EXERCISE TOLERANCE		ABILITY TO WORK	
				BEFORE	AFTER	BE FORE	AFTER
No Anginal Pain at Rest							
D M	41	Oil field worker	None	150 ft	Up to 10 miles	No	8 hr
C A	40	Machinist	Following severe exer cise only	150 ft	½ mile	No	16 hr
A M	34	Grocer	Slight	50 ft	¼ mile	No	Works, and rides horseback
G A.	16	Farmer	None	200 ft	Ploughed 1,600 acres	No	Working
L G M	14	Physician	Yes	50 ft	Was 1 mile Influenza and extra systoles limited	No	No
Angina Status							
Three patients died in first 62 hours postoperatively							
Died Recently							
F S *	37	Executive	Yes	100 ft	100 ft or less	No	No
S E †	22	Electrician	Occasional	50 ft	¼ mile	No	8 hr

*L. S. One failure in group Died of aplastic anemia—artery blocked
†S. E. Was returning from work pushed car out of snow and died of rupture of old
scar interventricular septum

TABLE XII. SUBSEQUENT USE OF CORONARY ARTERY Bypass FOLLOWING 1 TO 10 MONTHS

AGE	SEX	AGE	OCCUPATION	TIME DISABLED (MONTHS)	EXERCISE TOLERANCE	PAIN AT REST	SMOKING	HYPERLIPIDEMIA	TYPICAL INFARCTION	
					No	Exhausted Pain at Rest				
C H	M	75	Engineer	17	11 steps	Yes	Yes	Yes	Posterior	
M B	M	70	Electrician	18	30 steps	Yes	Yes	Yes	Ant. septal	
W B	M	73	Ship engineer	21	30 steps	No	Yes	No	Posterior	
B P	M	72	Cook	9	20 steps	Yes	No	No	Posterior	
P O	M	70	Carpenter	Working, 10 nitros	16 steps	No	No	No	Posterior	
C H	M	74	Accountant	Working	16 steps	No	No	Yes	Operation not completed	
G I	M	70	Air pilot	6	20 steps	Yes	No	Yes	Posterior	
W B	M	74	Shipman	Working	19 steps	No	No	No	Posterior	
I J H	M	73	Oilman	12	11 steps	Yes	Yes	Yes	Post. wall	
J H	M	70	Metal worker	17	10 steps	No	No	Yes	Large anterior	
C I	M	73	Tractor rep	12	15 steps	No	No	Yes	Ant. posterior	
B B	M	75	Mechanic	1	10 steps	No	No	No	Posterior	
C I	M	71	Farman	sedentary	No pain	No	No	Yes	Posterior	
I I	M	70	Mechanic	17	4 block	No	No	Yes	Posterior	
C S	M	72	Carpenter	1 years	1 block	Yes	No	No	Posterior	
A S	M	75	Shipman	sedentary	27 steps	No	No	No	None	
P K	M	75	Hotel man	Working	1 block	No	No	No	None	
C A	M	70	Air craft	2 months	900 s/sk	Yes	Yes	No	None	Septal anterior
P A H	M	71	Act. amputee	6 months	900 s/sk	Yes	Yes	Yes	Ant. lateral	
				9 months	900 s/sk	Yes	Yes	Yes	Posterior	

TABLE XIII RESULTS SECOND GROUP 19 CASES FOLLOWED 1 TO 10 MONTHS

NAME	TIME SINCE OP (MONTHS)	OCCUPATION	ANGINAL PAIN	EXERCISE TO FRANCE		ABILITY TO WORK	
				BEFORE	AFTER	BEFORE	AFTER
C Hill	11	Engineer	Yes	11 steps	Not tested, improved	No	Not working
M B	9	Rail sec man	Occasional	30 steps	1/2 mile	No	Part time
W Hart	6	Sta eng	No	30 steps	3-4 miles	No	Working
B Phr	6	Cook	Yes	29 steps	Few blocks	No	No
P Ol	1	Carman	Occasional	16 steps	Improved, has reduced nitros from 10 to 0	Yes	Full day
G T	7	Air pilot	No	20 steps	1-2 miles	No	Full day
W B	11	Salesman	No	49 steps	Not known	Yes	Part time
L Th	4	Oilman	No	11 steps	Not known	No	Part time
J Han	1	Metal worker	No	14 steps	1 mile	No	Full time
C Ess	1	Tractor rep	No	15 steps	Marked improv	No	Full day
R Han	1	Mechanic	No	19 steps	Complication	empyema, still draining	
C Tol	1	Ironman	No	1 block	Unknown	No	Working
F Trev	2	Merchant	No	1 block	25 yd	Seldom	Working
C Shep	1	Carpenter	No	25 steps	Not known	Seldom	Part time
<i>Angina Status</i>							
T Kl	5	Hotel man	?	None	Not known	No	Not known
G Adams	5	Aircraft	Yes	None	1 mile	No	Working
Par	5	Vet Symes amp	Yes	None	50% improved	No	Walking
A Sto	5 1/2	Salesman	Died on operating table,		pulmonary edema	One death in 19 cases	

Twelve or 67 per cent are free of pain, with increased exercise tolerance. Three still have pain but are improved. One has severe pain with no improvement. One has not been followed. Thus of the 29 patients there were

Twenty-three patients without angina at rest. In this group there was one death or a mortality rate of 4.1 per cent.

Of the 6 patients with angina status 3 died in the early series giving a 50 per cent mortality.

The mortality rate for the entire group is 14 per cent.

There were 24 patients who survived, 23 of whom had the implant operation. Fifteen or 60 per cent are now completely free of pain. Seventeen or 73 per cent have returned to work after operation. Many are working 8 to 16 hours daily at hard physical labor.

SUMMARY

The clinical results of internal mammary artery implantation in the treatment of coronary artery insufficiency are most encouraging.

About 70 per cent of our patients are markedly improved and have returned to work.

For cases without angina at rest the mortality rate is low—4.3 per cent.

In the experimental animal the implanted internal mammary artery has been shown to send out large branches in the ventricular myocardium in 71 per cent. These branches are large enough to carry a supply of fresh blood into the heart at a rate as high as 51 c.c. per minute.

The pericardial fat pads are used as grafts applied to the surface of the left ventricle to supplement the internal mammary artery revascularization of the left ventricle.

The results on our human patients are beginning to parallel very closely the frequency of mammary coronary anastomosis demonstrated in our animal experiments.

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DISCUSSION

DR W B NEPTUNE, Boston Mass—In the Overholt group we have been interested in the problem also and in the past have done several of these procedures on clinical cases. However, I should like to present some of my own observations.

(Slide) Of 18 experimental procedures done on normal animals, 2 were found to be thrombosed, 5 showed no anastomosis, 5 supplied a small capillary bed, 6 went to the coronary system and, of these, 3 went to the arteries and three to the veins. If we make a section through the transplanted artery, note the intense intimal proliferation with decreased cross diameter of the lumen. This is nine months after surgery and I think Dr Vineberg's sections showed similar findings. This has been uniform in our experiments.

Our anatomic studies are based upon injections in living animals, using a 4 per cent gelatin solution, containing a colloidal suspension of carbon particles, 27 μ in diameter, which has been suitable for the demonstration of myocardial capillary bed. This has been injected through the internal mammary artery at constant body temperature and at the same pressure as found in the arch of the aorta. For the sake of this discussion we will forget all cases except the most ideal and those are the only ones I wish to speak of.

Grossly we have found that the carbon particles are limited to the anterior surface of the heart and around the myocardial tunnel itself. If we make a tangential section through this area we note that the carbon particles again are right around the myocardial tunnel in Zone I and in Zone II (20 mm from the tunnel) we do not see much. This is a low power field of the entire section of the heart showing the transplanted artery. If we

make a high power section through Zone I, right around the tunnel itself, we see an excellent demonstration of the myocardial capillary bed, excellent filling, no question about it. In Zone II which is 20 mm away, I do not think we can demonstrate any evidence of myocardial capillary filling. If we make a cleared section of this same area after the Spalteholz technique we cannot demonstrate any evidence of vascular pattern.

Just to let you know that we can demonstrate capillary filling if we make a cleared section of a normal heart injected through the coronary artery this is the normal pattern which we can demonstrate. These studies to us would suggest that revascularization by means of a transplanted artery in a myocardial tunnel, per se, offers little in the way of flow or area supplied.

DR A R HENDERSON Ashbury Park, N. J.—Much impressed with the results reported by Drs Vineberg, Samuel Thompson Beck and others during the past few years we have repeated the various methods designed to increase collateral flow of blood to the heart in the experimental laboratory. Although a considerable portion of the dog's heart could be vascularized by any of the procedures advocated (e.g., pericardial insufflation with tale, internal mammary artery implantation, arterialization of the coronary sinus), we were very much disgusted with the unreliability of any one procedure to function as a revascularization phenomenon. Since coronary artery disease usually involves the entire coronary system it was felt that in the surgical treatment of human cases no one procedure should be relied upon which would most likely vascularize only a portion of the failing coronary circulation.

For that reason in treating patients surgically we have subjected them to combinations of revascularization procedures. Thus in one operation, 20 patients have had internal mammary implantations, pericardial poudrage and left upper dorsal sympathectomy. Sympathectomy (T₁, T₂) was performed not to relieve the patient of angina but because we have been very much impressed with recent work on the role of sympathetic secretory mechanisms (particularly the over-secretion of norepinephrine) in producing an oxygen wasting type of myocardial metabolism. Denervation in these cases, probably reduces the oxygen requirements of the myocardium thus reducing demands upon the coronary flow.

Of the 20 patients thus far operated upon within the past fourteen months only one has died. There have been no operative deaths. The one patient succumbed to a massive occlusion eight days postoperatively. It is hardly fair to judge the results of such combined therapy at this time but thus far the clinical results are gratifying. Dr Vineberg is certainly to be congratulated for his persistent efforts and his contribution to the surgical management of coronary artery disease.

DR A D CHECCA Newark, N. J.—We were first introduced to this surgery of coronary artery disease in 1951. At that time our mentors were Drs Bailey and Beck. We started with the Beck procedure that is the Beck No. 2 which as you know is a shunt operation and we had some very interesting observations on that and on the other operations. We have used all types to date in 26 patients—the Beck No. 2, the Beck No. 1, the Thompson operation and also the Vineberg. Clinically those patients who survived all seem to be better. An observation on the Beck procedure I think is of importance to this study. One of our patients after two years developed severe edema and cardiac failure. Autopsy showed that his shunt was now pouring 30 per cent of the blood volume through the shunt into his right auricle. The edema was uncontrollable by medical means, and he died after 10 days of operation in spite of the severe edema and tied off the coronary shunt at the time of autopsy. We feel that the coronary venous system had thrombosed and after he had all the shunt circulation to pour into the right auricle. After the shunt was tied off the edema completely disappeared within 48 hours. This patient is now doing very well. I am a member of the American Surgical Patients Club as well and very active in the club.

Of the 26 patients who have had Beck No. 2 procedures, 12 are still alive and well. Of the eight who had Beck No. 2 procedures, 12 are still alive and well. Of the eight who had Beck No. 2 procedures, 12 are still alive and well.

after the eighth day the aorta blew out and at autopsy there was absolutely no evidence of healing. The second patient died of cardiac arrest several hours after operation. A massage procedure was undertaken for three hours, the patient had no evidence of neurologic damage, the heart returned to function for no longer than 60 to 80 seconds at a time under massage and stimulation and, finally, after three hours we decided that the game was up.

We had two Vineberg operations with no mortality, three Thompson procedures with one death. We use the Thompson operation on cases with status angina, and feel that the procedure can be done more swiftly without much stress on the patient and is well tolerated.

DR ALFRED GOLDMAN, Beverly Hills, Calif—About a year and a half ago we developed the concept that perhaps some beta radiation might affect the ischemic heart. This concept was based upon the observations that radon ointments and ointments made with P 32 are capable of stimulating capillary ingrowth in chronic ulcerations associated with arteriosclerosis. The problem was to test this in dogs, and I would like to present some preliminary observations.

We used approximately 83 dogs. The largest branch of the anterior descending artery was ligated and at the same time 3 mc of colloidal radioactive chromic phosphate was instilled into the pericardium. After similar anterior coronary ligation in the control animals the same volume 6 c.c. of nonradioactive colloidal phosphate was instilled in the pericardium and the pericardium was closed tightly. The complete results of this procedure I cannot give at this time except to say that the dogs survived this procedure, grossly scarred infarcts which were vascular at the time of sacrifice were observed. We still have some 30 animals in this group to sacrifice, and this is a preliminary report.

(Slide) This is the section of the myocardium from a dog that was sacrificed at 64 days, and you will see the large infarct with ingrowth of capillaries and small vessels. This is the autoradiograph which shows the activity of the radioactive phosphate tending to localize on the epicardial surface and over the area of infarction.

(Slide) This is a microphotograph of the same animal showing vascularization in the infarcted area.

I simply present this as a different method, perhaps a different approach to the problem of revascularization of the ischemic heart.

DR IVAN D. BARONOFKY, Minneapolis, Minn—I should like to thank you for the privilege of the floor. Dr. Vineberg is to be congratulated on his ideas and results. Yesterday we heard Dr. Kev present a paper on the use of a jejunal transplant to the heart in order to revascularize the heart. Independently, my resident, Dr. Joseph Sprafka, and I, at the Ancker Hospital in St. Paul, have been using a somewhat similar procedure. We have used a defunctionated Roux en Y loop of jejunum or ileum and attached it to the heart. We have also used a flap of stomach muscularis.

(Slide) This shows the Roux en Y loop of intestine brought up to the diaphragm. The serosa of the defunctionated loop is stripped off for a good distance. This is very easily accomplished.

(Slide) Here is the loop being attached to the heart. The epicardium has been stripped off the heart using a very easy maneuver. The gut must be placed over the branches of the anterior descending coronary artery.

(Slide) Here is an injected specimen showing the gut attached to the heart. The mesenteric artery has been injected and, as you see, the entire coronary system has been visualized by the injection of the mesenteric vessel.

(Slide) Here is another injected specimen in which the anterior descending artery has been cut off and, as you can see, there is an excellent filling of the coronary system by the injection of the mesenteric artery.

We have done one human case recently, and it is much too early to arrive at any conclusion.

DR SAMUEL A. THOMPSON, New York, N. Y.—There are two general principles involved in myocardial revascularization: first the use of some form of extracardiac anastomosis and, second, the stimulation and further development of the residual myocardial circulation. Dr. Vineberg has very beautifully demonstrated the use of an extracardiac anastomosis. We do not feel that the potentialities of the residual myocardial circulation have been sufficiently emphasized. This residual myocardial circulation comes partly from the coronary system and partly from small vessels around the pulmonary artery, around the orifices of the superior and inferior venae cavae and from the pericardium and the aorta at the base of the heart.

This residual circulation is able to furnish a tremendous collateral supply under proper stimulation, and it may be true that the greatest good effect of all the operative procedures comes from this source of supply rather than from the extracardiac source. For sixteen years we have been using a simple but very effective method in the treatment of coronary insufficiency which utilizes the stimulation of the residual myocardial circulation plus the advantage of an extracardiac anastomosis and tissue graft. We have done the operation in 134 cases. The operation is known as cardiopexy, sometimes called "The Talcum Powder Heart Operation," and occasionally I have been asked "What fragrance do you prefer?"

The operation is done through a small anterior incision over the fifth cartilage; the pericardial sac is opened and all the pericardial fluid is removed. The surface of the heart is completely covered with magnesium silicate powder, some 2 to 6 teaspoons by volume and 4 to 12 Gm. by weight. We do not believe it is necessary to scarify or attempt to remove the epicardium as the silicate powder is a very strong irritant and produces a violent inflammatory reaction. This reaction is sustained in some form for the lifetime of that patient. The reaction has a recognizable pattern of behavior and a predictable end result. The simplicity of the operation with the low mortality has much to recommend it. We believe the end results are comparable to any method used.

We have approximately 10 per cent of poor results. Poor results mean anything less than 50 per cent improvement. The other 90 per cent of patients are improved more than 50 per cent. We have 40 per cent who are more or less normal. Normal means no medication and no restriction in physical activity.

DR. W. G. BIGELOW, Toronto, Ontario.—Thank you for the privilege of discussing Dr. Vineberg's paper. All who have studied this problem are well aware of the difficulties of assessing benefits from any attempt to revascularize the heart both in the experimental animal and in human cases. However, because of Dr. Vineberg's fine experimental results we were tempted to investigate the problem in our own laboratory. We have not seriously studied the apparent protection this operation affords to coronary ligation but rather have turned our attention to a study of the fate of the implanted artery. We examined rather carefully three internal mammary transplants in animals nine months after their insertion and after anterior descending branch ligation. It was the injection results and the pathologic picture in which we were interested.

(Slide.) We got good injection studies in each. The dye entered the coronary artery and not the vein in all three.

(Slide.) What we were interested in was the principle which Dr. Vineberg was following. It did not seem credible to us that one could put an artery in an organ and have that artery branch out and revascularize the organ. Following Dr. Vineberg's technique, but all collateral branches were cut and allowed to bleed vigorously just before the artery was drawn into the tunnel.

A similar pathologic picture and a similar result from injection has been obtained in each animal. Dr Ritchie has pointed out that the vulnerable area for the implanted area is where it enters the heart at the level of the epicardium. In recent studies of coronary artery disease, that has been proved to be the area where sclerotic changes are most likely to occur. The intramural section of the artery is usually well protected and shows little change. I am afraid that this area may show progressive narrowing over a longer period and be exposed to the danger of thrombosis.

It is a very interesting fundamental phenomenon—the concept of putting a major artery in an organ and having it supply the organ with branches of that size. It warrants more study.

DR CHARLES B. RIPSTEIN, Brooklyn, N. Y.—I think Dr Vineberg is to be commended on his excellent experimental work, but I would like to interject a word of caution on the wide application of any of these procedures to human material. In 1948, I published a review of all the surgical procedures then being used for coronary artery disease and came to the rather reluctant conclusion that none of them really offered any lasting benefit. I am presently completing another such review and am again, reluctantly, of course, coming to the same conclusion.

DR ARTHUR M. VINEBERG (Closing).—The results reported by Dr Neptune on a small series of animals subjected to internal mammary artery implant are not surprising. They are exactly the same type of results that my resident or research Fellow obtains in the first few months when working with us. For example, this year Dr William Buller, who started to do internal mammary artery implants in July, found that 80 per cent of his arteries were blocked in the first two months of his experience. After he had learned to implant the artery according to a technique which has been laboriously worked out over a number of years in this center, 86 per cent of his arteries remained open.

For those who find internal mammary artery blocks in a high percentage of experimental animals, I would suggest that they visit our laboratory, where our experimental Fellow will be pleased to show them the various technical points we have learned. Dr William Bigelow did this two years ago, and has conveyed to me the fact that practically all his implanted mammary arteries in animals remained open. Dr Bigelow had added one other feature to the technique—namely, that of wrapping the internal mammary artery in the medial pericardial fat pad. This appears to prevent the artery from angulating and from adhering by scar to the neighboring structures.

The injection technique used by Dr Neptune is most difficult to evaluate as the injection mass was introduced into the internal mammary artery while the heart was beating. It is well known that in order to demonstrate anastomotic channels in the ventricles of either human or animal hearts, it is necessary to place the hearts on ice for at least twenty-four hours in order to get rid of the spastic elements that occur immediately after death. Many others have confirmed the frequency of internal mammary artery anastomosis, and have found that the facts as given by us are approximately the true situation.

We have found that the frequency with which the internal mammary artery joins up with the arterioles in the left ventricle is dependent upon the condition of the heart at the time the artery is implanted. For example, when placed in normal hearts one cannot expect more than approximately 46 per cent anastomoses. When placed in an ischemic heart the anastomosis rate is increased to around 76 per cent. The internal mammary artery will stay open without necessarily giving out large branches, in more than 86 per cent of all implants when the proper technique is followed.

In those animals in which coronary artery insufficiency had been produced by wrapping Cellophane around the anterior descending branch of the left coronary, the implanted internal mammary arteries remained open in every instance.

There is no question that there will be some intimal proliferation; however, this usually occurs distal to the open sixth intercostal artery. The portion distal to the sixth intercostal is not in use and therefore tends to block off by intimal proliferation just as the ductus arteriosus or other vessels in the human body do when their presence is no longer required.

ANATOMIC, PHYSIOLOGIC, AND SURGICAL CONSIDERATIONS IN REPAIR OF INTERATRIAL COMMUNICATIONS IN MAN

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OPERATIVE repair of atrial septal defects is now feasible, largely because of the efforts of Cohn,¹ Swan,² Murray,³ Bailey and associates,⁴ Lewis and Taubert,⁵ Gross and co-workers,⁶ and others. It is important, however, to clarify the indications for operation, to select the best methods of repair, and objectively to assess the results. The necessity for exact diagnosis has resulted in a clearer concept of the hemodynamic aspect of the condition, but certain problems require further elucidation. Our experience to date with the anatomic, physiologic, and surgical aspects of interatrial communications is presented.

ANATOMIC VARIANTS IN ATRIAL SEPTAL DEFECT

Problems of surgical repair demand an exact knowledge of the anatomic variants of interatrial communications. A classification of these communications is presented in Table I.

TABLE I CLASSIFICATION OF INTERATRIAL COMMUNICATIONS

I	Normal interatrial communication—valvular competent patent foramen ovale
II	Abnormal interatrial communications
A	Valvular incompetent patent foramen ovale
1	With complete posterior septal rim
2	With absent posterior rim and "secondary" anomalous pulmonary venous connection of right lung
3	With associated congenital anomalous pulmonary venous connections
B	Defects of the lower portion of the atrial septum as part of persistent common atrioventricular canal
C	Communications of unusual location or nature

Valvular Competent Patent Foramen Ovale—An interatrial communication is present in about 25 per cent of normal people, the so-called valvular-competent patent foramen ovale. This has been termed the "probe-patent" foramen ovale, implying small size, but in adults the tract may measure 1 cm or more in diameter. In normal persons the pressure in the left atrium exceeds that in the right atrium, hence the valvular flap is kept closed. Under these

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circumstances, left-to-right shunts cannot occur. In congenital or acquired conditions wherein the right atrial pressure exceeds that in the left atrium, right-to-left shunts develop across these normal interatrial communications.

Valvular-Incompetent Patent Foramen Ovale—A valvular-incompetent patent foramen ovale is present in some persons. This is an abnormal inter-



Fig 1—Normal heart viewed from front showing proximity of orifices of right pulmonary veins (black probes) to the atrial septum. Orifices of left pulmonary veins are far removed from the septum. This anatomic arrangement permits preferential shunting of blood from right lung across atrial septal defects. Orifice of inferior vena cava (IVC) lies close to fossa ovalis. CS = Coronary sinus.

atrial communication that usually is crescentic in shape and related to an inadequate length of the flap of the original septum primum. However, such defects do vary in size and exact location. Occasionally, instead of being a clean hole, such a defect may have lacy strands of tissue traversing it. The orifices of the right pulmonary veins in the posterior wall of the left atrium are near the atrial septum and thus are in close proximity to an atrial septal defect (Fig 1). The orifices of the left pulmonary veins are considerably removed from the septum, since they connect to the left lateral wall of the atrium.

Many atrial septal defects of this type, although large, possess a complete rim of septal remnant. Some, located near the inferior or superior vena cava, are not bounded by septal remnant at the orifice in question. A special problem is posed by large atrial septal defects of this type in which no rim of septal remnant exists posteriorly. The orifices of the right pulmonary veins, which normally lie near the septum in the posterior wall of the left atrium, in these cases lie to the right of the plane of the septum.⁴ This situation may be described as an anomalous connection of the veins of the right lung to the right atrium. The anatomic malformation could be termed atrial septal defect, and "secondary" anomalous pulmonary venous connection.

A true congenital anomalous pulmonary venous connection of the right or the left lung may be associated with this type of atrial septal defect. In contrast to the afore-mentioned "secondary" anomalous connection, congenital anomalous pulmonary venous connections are found at sites predictable on an embryologic basis. An important variant of this type is congenital anomalous connection of the right pulmonary veins to the superior vena cava and cephalic portion of the right atrium.

Defects of the Lower Portion of the Atrial Septum—Interatrial communications may lie just above the rings of the mitral and tricuspid valves. In such cases the rings and the valvular leaflets themselves may be cleft and the defect may extend down to involve the upper portion of the ventricular septum, this condition is called "persistent common atrioventricular canal."

Communications of Unusual Location or Nature—Rare communications such as by way of a coronary sinus with a connection to the left atrium, will not be considered.

PHYSIOLOGIC STUDIES

Fortunately these anatomic variations cause specific physiologic findings that frequently permit their preoperative identification. Study by cardiac catheterization in all patients clinically diagnosed as having atrial septal defect and being considered for operation is necessary for precise diagnosis and for localization of the defect.

Determination of intracardiac pressure and oxygen saturation and calculations of systemic and pulmonary blood flow in this study were made by methods previously described from this laboratory.¹⁰ Dilution curves of T 1824 were recorded in the arterial system by means of cuvette and ear oximeters after injection of the dye into chambers of the heart and great vessels during cardiac catheterization.¹⁰ The contours of such dilution curves are largely determined by the characteristics of flow through the heart and lungs and are distorted in a specific manner by flow through abnormal vascular communications.^{11, 12} In general dilution curves recorded from patients who have defects characterized by left to right shunts have lower peaks of amplitude than do curves recorded from normal persons, while the declining concentration of dye after the peak is disproportionately prolonged due to repetitive recirculation of a portion of the dye through the heart and lungs with each circulation.

Physiologic data from 53 patients who had atrial septal defects and who were studied in the cardiac catheterization laboratory at the Mayo Clinic have been summarized by Weidman and associates¹³ (Fig 2), these data are in essential agreement with the findings of most other workers. They indicate that systemic flow is typically normal in this condition in the absence of cardiac failure, but that pulmonary blood flow is characteristically increased. In most instances, the pulmonary arterial pressure is a little above the normal range.

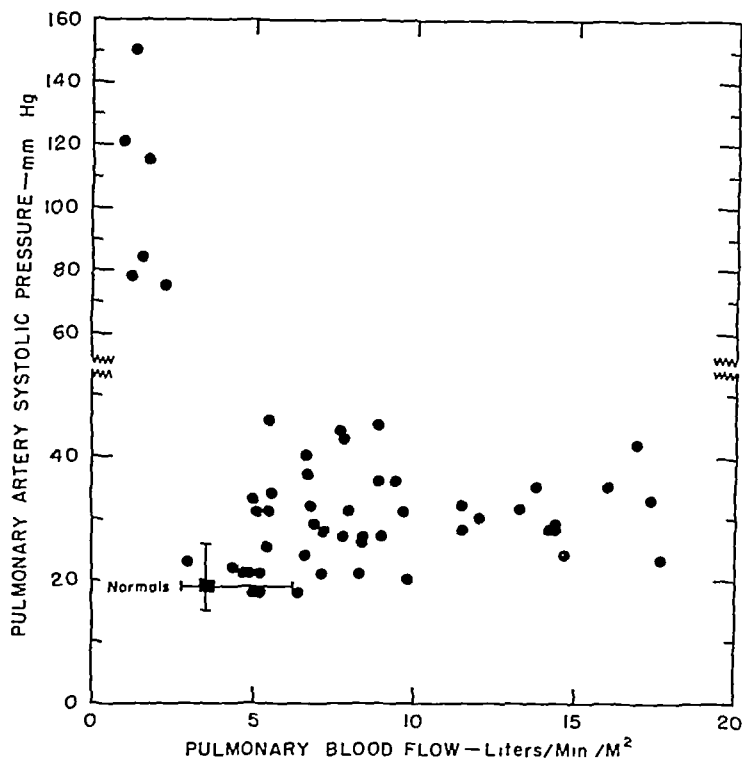


Fig. 2—Relation of pulmonary arterial systolic pressure to pulmonary arterial blood flow in 53 patients who had atrial septal defects.¹³ The average value for 23 normal persons is represented by the solid square; the lines extending from this square show the ranges of normal. A moderate increase in pulmonary systolic pressure that may not be correlated with increasing flow is usual in patients who have atrial septal defects. Note that pulmonary flow is actually reduced in patients who have pronounced increase of pulmonary arterial pressure, an exception being Case 11 studied after these data were compiled.

In uncomplicated atrial septal defect, the flow across the defect is predominantly from left to right and is often of great magnitude. By means of the indicator-dilution technique it has been possible to demonstrate preferential flow of blood through the defect from the right lung in cases of defects of the type characterized by a valvular-incompetent patent foramen ovale without either "secondary" or congenital anomalous pulmonary venous connection.¹⁴ Thus, in the usual case of atrial septal defect, most of the blood streaming into the left atrium from the left pulmonary veins goes through the

adjacent mitral valve into the left ventricle while a smaller amount is shunted across the septal defect. In contrast, a greater proportion of the blood entering the left atrium from the right pulmonary veins flows across the anatomically adjacent defect in the atrial septum and a smaller amount continues

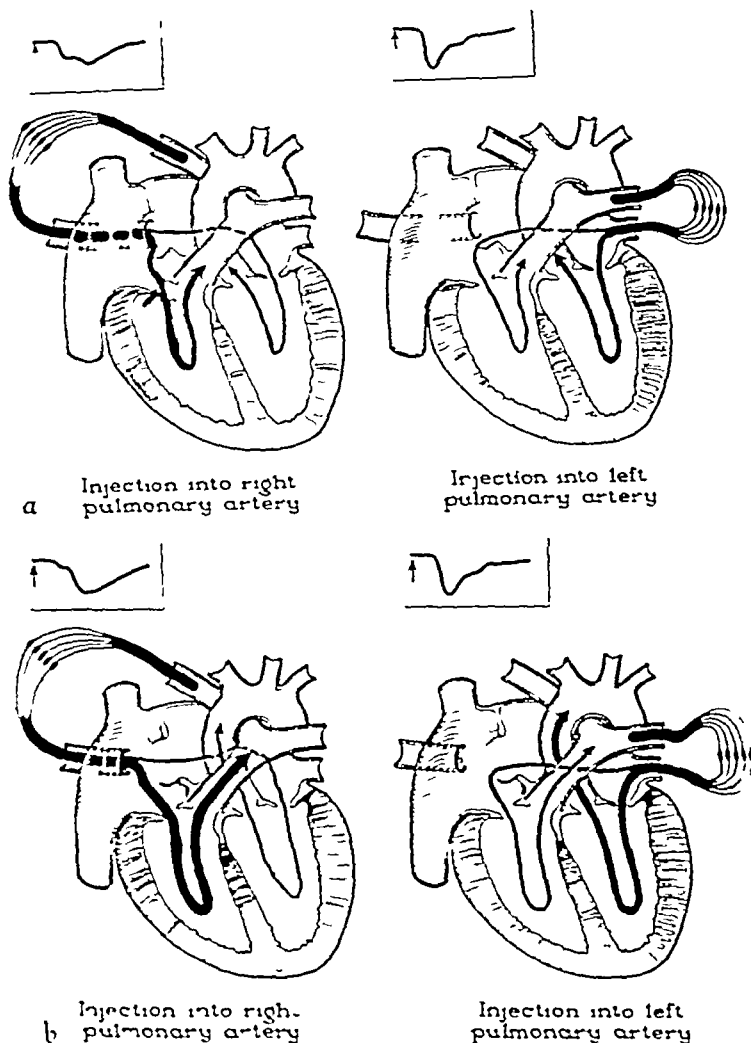


Fig. 1.—Left to right shunts through atrial septal defects: diagrammatic representation of dye dilution curves taken by dye injected into the right and left pulmonary arteries. A solid circle indicates the site of injection; the typical contour of the resultant dye dilution curve is indicated by each diagram. The instant of dye injection is indicated by a vertical arrow. (a) Dye injected into the right pulmonary artery: the greater proportion of it usually flows into the right atrium through the atrial septal defect, presumably because of the anatomical proximity of the defect and illustrated in Fig. 1. When dye is injected into the left pulmonary artery, the greater proportion passes through the orifice of the mitral valve and enters the systemic circulation. The dye dilution curve is different in that when dye is injected into the left pulmonary artery, the initial deflection of the curve is small because of the relatively small contribution of blood from the right lung to the systemic circulation; the greater contribution of blood from the left lung to the systemic circulation through the lungs. (b) Circulatory path taken by dye injected into the left pulmonary artery: the dye can be traced through the pulmonary veins into the left atrium and then through the atrial septal defect into the right pulmonary artery, and then through the right atrium to the systemic circulation. Almost all of the dye injected into the right pulmonary artery passes through the right atrium to the systemic circulation and then to the systemic circulation.

across the left atrium to pass through the mitral valve (Fig 3, a) The disposition of the veins in the posterior wall of the left atrium, previously mentioned offers ready explanation for this finding

In cases of atrial septal defect with absence of the posterior rim associated with "secondary" anomalous pulmonary venous connection of the right lung, indicator-dilution curves recorded after injection of dye into the right pulmonary artery indicate that an extremely small amount of blood from the right lung goes to the left atrium and that almost all of it traverses the right atrium to pass through the tricuspid valve and recirculate through the lungs (Fig 3, b) In instances of associated congenital anomalous pulmonary venous connection, the dilution curves recorded after injection of dye into right or left pulmonary arteries indicate that practically all the blood from the anomalously connected lung passes into the right atrium and thence again to the pulmonary circulation (Fig 4, a)

Although the over-all shunt in most cases of atrial septal defect is from left to right, there is frequently a small right-to-left shunt of blood entering the heart from the inferior vena cava,¹⁵ which lies close to the defect This has been demonstrated by injection of T-1824 into the inferior vena cava In certain cases, a small right-to-left shunt also occurs from the superior vena cava (Fig 4, b) which lies relatively far from the defect

A separate category consists of cases in which there is predominantly right-to-left flow across an interatrial communication with consequent cyanosis This group usually includes atrial septal defect with severe pulmonary hypertension atrial septal defect with pulmonary stenosis, and atrial septal defect with Ebstein's malformation of the tricuspid valve

SELECTION OF PATIENTS FOR OPERATION

Certain broad indications for operation in patients who have atrial septal defect are recognized in spite of the small numbers of patients who have been studied and operated on Conclusions in this regard must be considered tentative and indubitably will require revision in the light of further experience

Patients who have a left-to-right shunt in excess of 50 per cent of the pulmonary blood flow are considered as possible candidates for operation Surgical intervention appears particularly indicated when symptoms are present or when the shunt is even more excessive

When the pulmonary arterial pressure is considerably increased, with systolic values greater than 80 mm Hg large left-to-right shunts are usually absent indeed in such instances either a mixed or a completely right-to-left shunt may be present In a case recently studied (Case 11 of this report) a large left-to-right shunt occurred in the presence of severe pulmonary hypertension Our experience in this case and the findings of Gross and Watkins¹⁶ suggest rather strongly that attempted closure of an atrial septal defect by the atrial-wall technique in the presence of pronounced pulmonary hypertension is accompanied by great risk to the patient

As already indicated atrial septal defects occur in association with pulmonary stenosis in some patients Usually the flow across these defects is

from right to left, but several cases of bidirectional or pure left-to-right shunts have been demonstrated.¹⁷ In the latter situation, consideration should be given to closure of the defect at the time of pulmonary valvotomy if the shunt is large.

Few data are available on which to decide as to the advisability of closure of interatrial communications in which the shunt is from right to left, with consequent cyanosis. In certain carefully selected cases, it may be beneficial to close such defects.

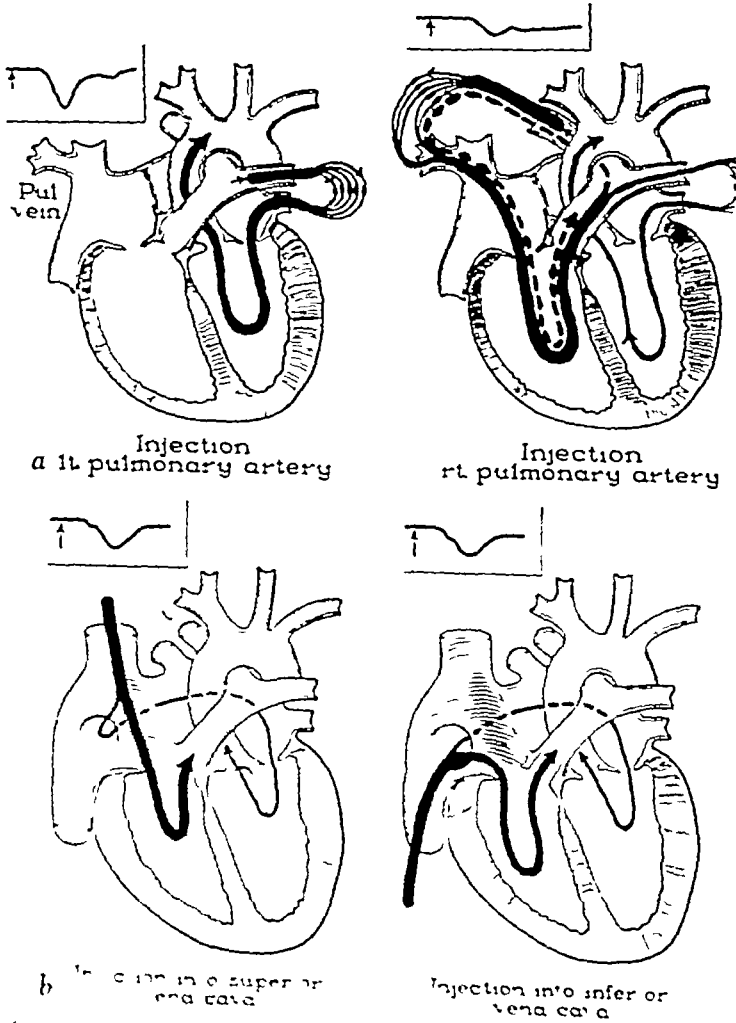


Fig. 1—Diagrammatic representation of path of dye injected into the right and left pulmonary arteries and into the superior and inferior vena cava. Noting the time of appearance of the dye in the right and left atria and the time of appearance of the dye in the right and left ventricles. In the right pulmonary artery, the dye appears first in the right atrium and then in the right ventricle. In the left pulmonary artery, the dye appears first in the left atrium and then in the left ventricle. In the superior vena cava, the dye appears first in the right atrium and then in the right ventricle. In the inferior vena cava, the dye appears first in the right atrium and then in the right ventricle. Noting the time of appearance of the dye in the right and left atria and the time of appearance of the dye in the right and left ventricles. In the right pulmonary artery, the dye appears first in the right atrium and then in the right ventricle. In the left pulmonary artery, the dye appears first in the left atrium and then in the left ventricle. In the superior vena cava, the dye appears first in the right atrium and then in the right ventricle. In the inferior vena cava, the dye appears first in the right atrium and then in the right ventricle.

OPERATIVE TECHNIQUES

Approach—After the introduction of the atrial well by Gross and associates,⁶ we abandoned an earlier experimental technique devised by one of us (Kirklin) and Donald and Gindlay.¹⁸ The well was used extensively in the laboratory and then in the surgical treatment of twelve patients who had atrial septal defects. This technique has proved to be a satisfactory method of approach for repair of these lesions. Since we have not utilized the other methods of approach that have been employed in humans,^{2, 4, 5, 19} we are not in a position to compare them with the technique employing the atrial well.

The thorax is opened through the right fifth interspace, after a long lateral incision is made. The atrial well is sutured in place with continuous silk sutures, after an interrupted suture is placed at either angle. Self-retaining retractors have not been placed inside the well. Two cubic centimeters of a 0.04 per cent solution of heparin is dripped into the well each minute. The clotting time of venous blood is more than one hour in most cases immediately after removal of the well, so 25 to 50 mg. of protamine is given slowly into a vein at that time. The well was tolerated by the heart without difficulty (except in one case to which reference is made later), accurate work may be done in the right atrium through it.

Method of Closure—In view of the results of an investigation of this problem in our laboratory^{18, 20} and the work of Bahnson and Baker,²¹ we have used thin pieces of polyvinyl sponge (ivalon) for closure of these defects. The sponge is sufficiently pliable to follow the contours of the septal defect after being properly sutured into place. Connective tissue from the septum invades the sponge to hold it firmly in place and the surfaces are rapidly endothelialized.²⁰

It is important that a large number of sutures, placed close together, be employed to attach the sponge to the remnant of the atrial septum (Fig 5, *a* and *b*). In regions where the septal rim is absent, such as occasionally occurs posteriorly or near the caval orifices, the sutures must be brought through the heart to the outside, as emphasized by Gross and co-workers,⁶ to be tied over small squares of sponge. Otherwise, there may not be a firm attachment of the sponge in such regions.

Special problems are encountered occasionally in placement of the sponge plug. Accurate preoperative studies are of great value in alerting the surgeon to these prior to operation. A persistent common atrioventricular canal was repaired in one case by anchoring a sponge plug in the defect by a number of sutures in the rim of atrial septum. Two sutures were placed along the top of the ventricular septum and one was put in the base of the septal leaflet of the tricuspid valve (Fig 6).

In cases of large atrial septal defects with absence of the posterior rim and with secondary anomalous pulmonary venous connection of the right lung in which this technique is used the polyvinyl sponge must be sutured into place so that its posterior aspect lies to the right of the venous orifices.⁶ Anteriorly, the sponge is made to curve to the left in such a manner that the caval orifices enter the atrium to the right of the sponge. The sutures must be tied outside the heart in the absence of septal remnants, as mentioned pre-

vously (Fig 7 *a* and *b*) In one instance (Case 10) of atrial septal defect with congenital anomalous pulmonary venous connection of the entire right lung into the lower superior vena cava and high right atrium, a repair was made by utilization of this same principle

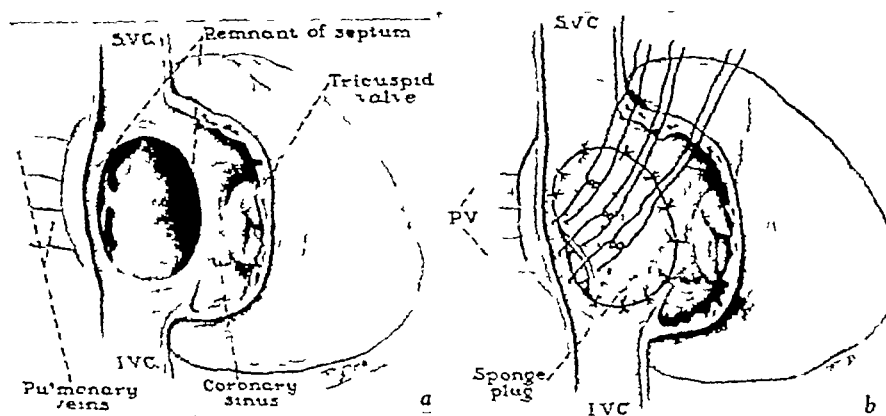


Fig 5—Large atrial septal defect (valvular-incompetent patent foramen ovale) *a* Note complete rim of septal remnant. *b* Closure of defect by plug of polyvinyl sponge sutured into place by interrupted silk sutures

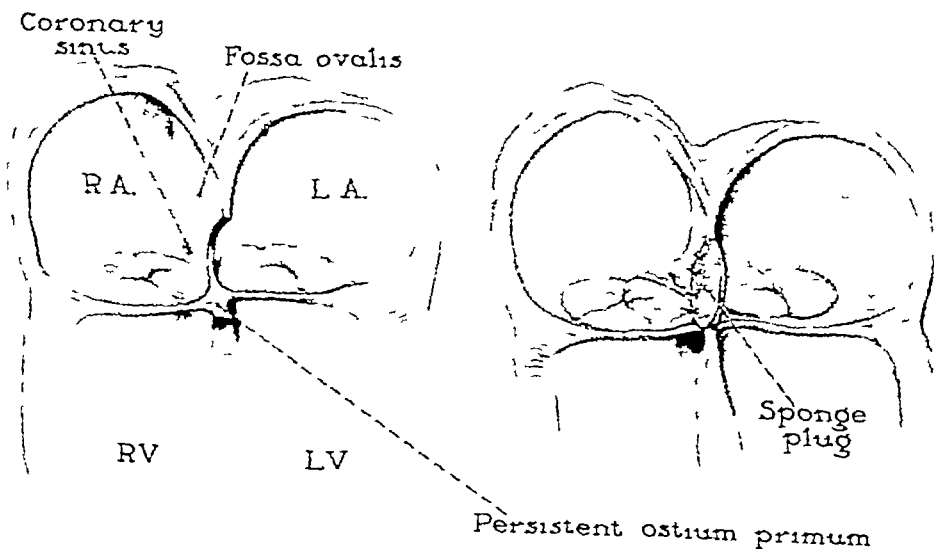


Fig 6—Atrial septal defect (partial persistent atrioventricular canal) The defect extends down so that its lower margin is the ventricular septum The defect was repaired by a sponge plug

RESULTS

A single death occurred in our previously mentioned group of twelve patients. The remaining eleven patients were alive and in good clinical condition when this article was written. The results are summarized in Table II.

TABLE II DATA ON 12 PATIENTS SURGICALLY TREATED FOR ATRIAL SEPTAL DEFECTS

DEFECT	CASE (NO IN SFRIES)	AGE (YR) AND SEX	PREOP SHUNT (PER CENT*)	POSTOPERATIVE STUDIES†		RESULTS
				INTERVAL POSTOP	FINDINGS	
Valvular competent patent foramen ovale with Ebstein's malformation of tricuspid valve	2	30 M	R L 25	3 wk	Residual 3 per cent R L shunt	Excellent
Valvular incompetent patent foramen ovale without anomalous pulmonary venous connection	3	27 F	I R 78	7 mo	Residual 33 per cent L R shunt also shown in dye dilution curve	Improved
	8	30 F	L R 70	2 mo	Very small residual L R shunt, dye dilution curve compatible with same	Excellent
	9	39 F	I R 71	Not yet studied		Operative death
	12	29 F	L R 75	Not yet studied		
	11‡	48 F	L R 80			
	5§	23 F	L R 88	Not yet studied		
	7	20 F	R L 16, L R moderate	Not yet studied		
Valvular incompetent patent foramen ovale with "secondary" anomalous pulmonary venous connection	1	43 F	L-R 80	14 mo	Residual 60 per cent L-R shunt (also shown in dye dilution curve)	Unsatisfactory
	4	22 M	L R 80	5 mo	No residual shunt, dye dilution curve normal	Excellent
Atrial septal defect with congenital anomalous pulmonary venous connection of right lung to superior vena cava and high right atrium	10	23 M	I R 67	6 mo	No residual shunt, dye dilution curve normal	Excellent
Persistent common atrioventricular canal	6	28 F	L-R 68	6 mo	No residual shunt, dye dilution curve normal	Excellent

*R L shunts = per cent of systemic flow L R shunts = per cent of pulmonary flow

†Cases 2 and 10 studied by dye dilution curves only Cases 1 3 4 and 8 studied by dye dilution curves and cardiac catheterization

‡Pulmonary hypertension also present

§Mitral stenosis also present

||Congenital pulmonic stenosis and L-R shunt across atrial septal defect also present

The death (Case 11) occurred in a patient who had a large atrial septal defect and severe pulmonary hypertension, with a pulmonary arterial systolic pressure of 120 mm Hg and an elevated right atrial pressure. Although it was

difficult to make the decision to operate, the fact that there was persistence of a large left-to-right shunt and that the patient's clinical status was serious made an attempt at surgical correction advisable. After application of the well and while the sutures were being inserted into the septal remnant, cardiac failure ensued and the patient died.

In Case 2, a valvular-competent patent foramen ovale and Ebstein's malformation of the tricuspid valve allowed a right-to-left shunt at the atrial level, with consequent cyanosis. The foramen ovale was closed by approximating the valvular flap with a single suture. Because of the right-to-left shunt in this patient before operation, the oxygen saturation of the systemic arterial blood was 86 per cent at rest, it decreased to 60 per cent on exercise.

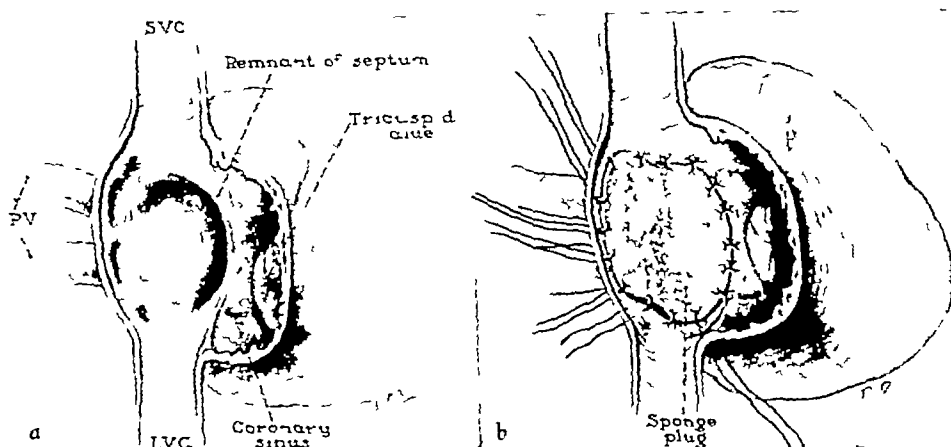


Fig 7—Large atrial septal defect (valvular-incompetent patent foramen ovale) with absent posterior rim and secondary anomalous pulmonary venous connection of right lung. *a* Note that the orifices of these veins are in a plane to the right of the orifices of the venae cavae. *b* Repair by a sponge plug so fashioned that it diverts the caval flow into the right atrium and flow from the right pulmonary vein into the left atrium.

After operation the systemic arterial oxygen saturation was 97 per cent at rest and 92 per cent on exercise. A dye-dilution curve indicated that a small (3 per cent) right-to-left shunt still persisted after operation. Symptomatic improvement has been pronounced.

In Cases 3, 5, 7, 8, 9, and 12, large (approximately 4 by 6 cm) atrial septal defects classed as valvular-incompetent patent foramen ovale without anomalous venous connection were closed with plugs of polyvinyl sponge. In Case 3, a good closure was considered to have been obtained at operation. At cardiac catheterization seven months later, a significant left-to-right shunt was still present, however, the magnitude of this shunt had been reduced from 78 to 33 per cent of pulmonary flow (Table III). Obviously, the sutures in this case must have given way at several points to allow separation of the sponge from a portion of the septal rim. One patient (Case 8) was studied two months after operation by the dye-dilution technique with injection into the superior vena cava and by cardiac catheterization. Closure was nearly complete, but a very small left-to-right shunt persisted. As the result of these

observations, even greater care has been taken in the other cases to anchor a large number of sutures securely in the septal remnant. Thus, in the remaining four cases of this group, secure closure was thought to have been obtained, but postoperative studies have not been made as yet. In one of these four cases, mitral valvotomy was done through the defect because of associated mitral stenosis. In another, pulmonic valvotomy was done during the same operation because of congenital valvular pulmonic stenosis with intact ventricular septum.

TABLE III CARDIAC CATHETERIZATION DATA BEFORE AND AFTER SURGICAL REPAIR OF ATRIAL SEPTAL DEFECT

	LOCATION	CASE 3		CASE 4	
		PREOPFRATIVE	POSTOPFRATIVE*	PREOPFRATIVE	POSTOPFRATIVE†
Pressure (mm Hg)	Right atrium	15/6	12/5	7/0	8/1
	Pulmonary artery	32/18	26/16	28/14	26/14
Blood flow‡	Systemic	3.0	4.2	3.1	3.6
	Pulmonary	13.8	6.3	14.2	3.6
Left to right shunt, per cent of pulmonary flow		78	33	79	0

*Seven months after operation

†Five months after operation

‡Liters per minute per square meter of body surface

In Case 6, uneventful convalescence occurred after what was interpreted as complete closure of a persistent common atrioventricular canal and of its associated septal defects. Postoperative studies were normal.

In Case 1, it was known at operation that incomplete correction of the defect had been obtained, this was confirmed by cardiac catheterization fourteen months after operation. In Case 4, a similar large atrial defect with absence of the posterior rim associated with a "secondary" anomalous pulmonary venous connection, was considered to have been completely repaired at operation. This was verified by cardiac catheterization five months later (Table III).

In Case 10, the contour of the postoperative dye dilution curve was entirely normal and no shunts were found 6 months postoperatively.

SUMMARY AND CONCLUSIONS

Surgical repair of abnormal interatrial communications demands exact knowledge of the anatomic variants of such anomalies. Precise study by cardiac catheterization and selective dye-injection techniques often allows accurate diagnosis and localization of these cardiac defects.

A study is presented of twelve patients who had surgical treatment of interatrial communications. Other cardiac anomalies were associated in some instances. The operative technique involved use of an atrial well and plugging of the defect with polyvinyl sponge. Only one death occurred in this series and that took place during operation on a patient who had severe pulmonary hypertension.

Tentative criteria are given for the selection of patients for surgical correction of interatrial septal defects.

ADDENDUM

We have now operated on 24 patients with atrial septal defect with predominantly left to right shunts. The only death in this group is the one already reported in this paper.

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DISCUSSION

DR WILLIAM L. JAMISON, Philadelphia, Pa.—I should like to give you our experience with 47 cases of atrial septal defect that we have closed at the Bailey Clinic (Slide). To palpate the defect in one case an ungloved finger is inserted through the auricular appendage. An anchoring suture is then placed to approximate the auricle to

the remnants of the septum. Next, a line of continuous suture is inserted following the first suture that was placed to anchor the auricle to the septum. Here the defect is completely closed. The auricle has been sutured to the remnants of the septum and the opening through which the finger is inserted is now closed. This creates a patch which closes the defect. This is the first case treated in this manner. The shunt was entirely closed, preoperatively it was 13.2 liters, postoperatively it was zero.

In cases where there are anomalous pulmonary veins the defect may be closed in a similar fashion. This particular idea was first suggested by Dr. Neptune. At that time he was interested in closing an atrial septal defect, but I expect now he will be spending some time with Dr. Vineberg.

In cases where the defect is very large, a passageway is created between the superior and the inferior vena cava to allow blood to get to the tricuspid valve.

The mortality in cases of ostium secundum has been 9 per cent. In ostium primum the mortality has been much higher, but we are lowering this mortality considerably in our continuing work.

We have found this technique applicable in all cases and very satisfactory.

DR. ROBERT E. GROSS, Boston, Mass.—I think that Dr. Kirklin and his associates have done a fine job in the study and care of their patients.

To date we have operated upon 30 cases of interatrial septal defects, with direct suture closure or by the onlay of a polyethylene sheet. Our approach at present is to explore the right auricle with the left index finger, if there is a posterior defect which is not too high or too low, one can place a straight row of stitches, which brings in the back wall of the auricle to the anterior edge of the septum, and get a closure effectively and quickly. If we happen to find an orifice which is not very large, and not too high or too low, we use the Bailey technique. We have had some good results with it. If we find a hole which is very high against the orifice of the superior vena cava, or low down near the tricuspid valve, we prefer to lay on a polyethylene sheet to cover the defect. To date, one half of our cases have been treated by the "well" approach, and the remaining one half by the other forms of closure just indicated.

We have abandoned the use of Pronestyl both before and during operation. We gave up Novocain in the pericardium long ago, because of the depressing effects on the general cardiac mechanism. There is, however, a place for Novocain. In some cases the auricle is quite irritable, and working on it may produce many extra systoles or a run of tachycardia; we have found that by swabbing 2 per cent Novocain on that part of the auricular wall on which we will be working (without allowing the fluid to run down onto the remainder of the heart and thereby depressing it), one can get rid of most of these disturbing irregularities. We have very little confidence in digitalis, but we insist upon quinidine. We feel this drug helps many of these hearts to get through this surgery with a minimum of disturbance. If, during operation, we find that a heart is flagging or weakening, we use rather liberally 5 or 6 cc. of 10 per cent calcium chloride directly into the heart, and 20 to 30 cc. of 50 per cent glucose. We have no proof that these help, but we have a strong feeling that they have prevented many catastrophes.

While some of the older patients have recovered promptly from this type of surgery, four or five patients in the late thirties or forties, in spite of the fact that they have had an excellent closure of the defect, have required long periods of rehabilitation to get back into acceptable cardiac status. These hearts are "tired," and it takes a good deal of treatment to get them into satisfactory condition again. In contrast, children or young adults who have closure of septal defects exhibit a prompt and satisfactory recovery. These points lead us to believe that all young patients who have large shunts (despite the fact that they may have no symptoms at the time) should be operated upon. If the right ventricular output is twice or more than that of the left ventricle we think operation should be undertaken.

DR. JAMES L. SOUTHWORTH, Knoxville, Tenn.—Dr. Kirklin has very properly emphasized the hemodynamic side of the problem, where more work will need to be done.

On the other hand, there has been much technical searching, I think, for a method of operation to close atrial defects. After hearing Professor Crafoord last fall my associate, Dr Dabbs, and I became interested in trying his method to close atrial defects, and we tried it in animals. The essential feature, as you know, is to dissect a groove between the two atria and sew this to the intraventricular septum.

(Film) This shows the beginning of a dissection between the right and left atria. The pulmonary vein is at the bottom of the picture and the tissue is held there by a forceps. It was surprising to me how easy it is in animals to dissect the two atria. It is possible to clear the superior vena cava very far behind and to see the aorta there. The finger is now within the atrium and the suture will be passed into the intraventricular septum under finger guidance. There the needle came out a little too high, was withdrawn, and came out in the proper place where the groove was dissected. The animal was sacrificed and left in the same position, the needle was passed again into the intraventricular septum.

None of the eight dogs we operated on had difficulties during the operation, all survived except the one we sacrificed. Thus we were encouraged to try this operation on a 32 year old woman who had a very large defect of the septum primum type, about 8 cm. in diameter, we had no difficulty in closing this defect, and the patient has made an uneventful recovery.

DR RALPH A. DETERLING, New York, N. Y.—I think the work presented here both by the essayist and the discussants is excellent. I want to bring up two points from our experience. We have been faced with operating on desperately ill children. In this group we have encountered either large septum primum defects in which there is just a little ridge between the two valves as shown in Dr Kirklin's last case, or else an atrioventricularis communis where a good sized defect is also present between the valves and in the upper portion of the ventricular septum. Although we have had poor results in these cases, they do need surgery. I think it is important to emphasize that some of the patients who most need surgery still pose a great technical problem.

I mention one case because of the difficulties we encountered. There was thought to be a mitral insufficiency in addition to a large atrial septal defect. The pressure in the right auricle was 44 mm. Hg. We could not use a rubber well, since the blood would come up over the top. We invaginated the atrial wall by the Bailey technique, and each suture hole spurted blood vigorously, requiring a total of about 3,000 c.c. of blood during the operation in this 9 year old girl.

DR FRANK L. GERBODE, San Francisco, Calif.—I would like to compliment Dr Kirklin on the excellence of his presentation and on the manner in which he closed these defects so successfully. After experimenting with the well technique in the laboratory for a time, my associates and I thought there must be a simpler way of closing the smaller defects. Therefore, about two years ago, we began exploring the possibility of utilizing the plane between the right pulmonary veins and right atrium as a means of obtaining posterior atrial wall for closure, believing it would be better to close the defect posteriorly than to oppose the anterior wall as suggested by Bailey. We found, as Dr Crafoord did, that one could pass sutures beneath the superior vena cava and beside the aorta, and thus close the defect posteriorly. However, there are some disadvantages in this method of placing the sutures. I would like to present another method for suturing which might be called the "hook" technique.

(Slide) Most of the infolding techniques involve pushing down the lateral or anterior atrial wall or auricular appendage to fill the defect. I have found that if a sharp hook with a slender shaft is passed through the lateral atrial wall and guided to the margin of the defect with the left index finger placed in the auricular appendage, one can engage the medial margin and pull it in apposition to the lateral margin and lateral atrial wall. An assistant can then hold the hook, with the margins thus held together, while a suture is passed through them from the outside. The first suture should be placed at the lower most part of the defect, and the closure made from below upward.

(Slide) This shows the use of the hook as an aid in the closure of a larger defect, where the lateral atrial wall is used as a circular patch. Again, the closure should start at the inferior margin, gradually approximating the edges in a circular manner.

This is a very simple method and should be useful for all defects save the very large ones. When combined with dissection of the posterior atrial plane a minimum of distortion results. It should be remembered, however, that great variations exist in atrial septal defects, and when the pulmonary venous return is abnormally placed, the dissection may not be possible.

DR WILLIAM H. MULLER, JR., Los Angeles, Calif.—I would like to compliment Dr. Kirklin on the very fine work he has presented here, and I would like to mention briefly our own experiences in dealing with this problem. We have used primarily the procedure developed by Dr. Gross, because it gives one an opportunity to explore the interior of the auricle leisurely. Furthermore, one is not faced with the problem of air emboli. We have been able to approximate the edges of the defect by direct suture in all instances except one, which was in a patient with a septum primum, here we employed a nylon plastic patch, but about three weeks later we had to remove it because she developed severe cardiac failure.

We have operated on 17 patients and have had 2 deaths—one a 4 year old patient who had been in severe failure and one a 30 year old patient with severe pulmonary hypertension. We think the most critical point in the operative procedure is when one opens the wall. We routinely use an indwelling cannula in the brachial artery to measure continuous direct pressures, often the pressure will drop to almost zero shortly after the clamps are removed from the auricle. Following the rapid introduction of blood through the cannula the pressure usually becomes elevated and the heart action becomes stable.

(Slide) This slide shows some of the physiologic studies which we make on these patients during operation, before and after the defect is closed. There has been a drop in pressure in the right auricle and a rise in pressure in the left auricle, as one would expect. There is also a diminished pressure in the pulmonary artery and a rise in pressure in the aorta. The oxygen saturation does not drop quite as much as one would expect in the right auricle, but these patients are receiving high concentrations of oxygen.

DR F. JOHN LEWIS, Minneapolis, Minn.—I should like to call attention to another variation in the anatomy of this complicated defect that we have encountered in closing them under direct vision with the aid of hypothermia.

(Slide) I think this variation is about as common as anomalous pulmonary venous drainage. Four of the last seven patients we have operated on have had it. It concerns the position of the valve of the inferior vena cava, which is normally located to the right of the vena cava. In this variation of the defect there is no lower rim, instead the valve of the inferior vena cava forms a false lower rim. If repaired improperly, blood from the inferior vena cava would enter the left atrium after closure.

(Slide) After the first stitch is placed and tied down, the rest of the repair is easily accomplished with a running stitch. In these cases, as well as in those with anomalous pulmonary venous drainage, we have found that simply stitching the edges together has been satisfactory, though in many cases it is very important to place the stitches in the right position to form new margins. Grafts have not been necessary, perhaps because when the heart is empty and collapsed the edges fall together satisfactorily.

Our experience with infants and young children has been the poorest we have had. We have operated on 16 patients whom we thought preoperatively had atrial septal defects. Three infants were found to have anomalies that we considered to be inoperable at the time. Two had complete AV canals and one had a ventricular septal defect. None of the 3 survived. Of the 13 remaining, one patient did not have his operation completed, although he recovered. Another patient had a septum primum defect and died three days postoperatively of complete heart block. Of the remaining 11 patients one died because of a technical error, leaving 10 successfully treated cases. Among the 12 patients who actually proved to have atrial septal defects, 4 had the type of anomaly just mentioned,

4 had anomalous pulmonary venous drainage and one had a septum primum defect. There were 2 with complicating mitral stenosis. Actually only 3 had what one would consider to be a simple hole between the two atria.

DR EARL H. WOOD (Closing). First I must admit I am a physiologist and not a thoracic or cardiac surgeon so it is obviously impossible for me to comment on the surgical considerations in this discussion.

I would like to emphasize that atrial septal defect is often a complicated condition. In the last seven years we have studied approximately 130 cases in which communications between the right and left atrium were demonstrated by the presence of abnormal shunts in one or the other direction. These cases were studied by what might be called special techniques—namely, rapid sampling via the catheter with the cuvette oximeter which allows identification, localization and accurate measurement of the magnitude of the left to right shunts, and by selective arterial dilution curves which allow identification, localization and accurate measurement of right to left shunts. Recently because of our interest in the surgical correction of this condition Dr W. H. Weidman and Dr H. J. C. Swan very carefully analyzed catheterization records in our laboratory from more than 100 cases; their results emphasize that this is not a simple condition. Of these cases only 60 were found in which the atrial septal defect was the only anatomic anomaly. So, certainly we agree with the other commentators in the conclusion that this is often not a simple defect.

Even when present as a single defect the problems of correction differ from many other cardiac conditions amenable to surgery, such as patent ductus arteriosus and mitral stenosis where the variation is only one of degree. In atrial septal defect not only is there a variation in the size and type of hole but also in the position of the defect in the septum and the relation of the septum to the pulmonary veins.

With special techniques, especially arterial dilution curves, I have been surprised and gratified at our success in being able to predict for Dr Kirklin the approximate size of the hole and its position in the septum and relation to the pulmonary veins. As you may know most patients with atrial septal defect show a small right to left shunt. If this is very predominant from the inferior vena cava and absent from the superior vena cava it is good evidence that the defect is low in the atrial septum in the fossa ovalis region where inferior vena cava blood impinges when emptying into the right atrium. Studies of the left to right shunt, particularly the degree of preferential flow from the right lung, allow prediction of the presence of an anomalous venous connection or of absence of the posterior rim of the septum. In the majority of cases we have been able to predict quite accurately for Dr Kirklin what he may expect to find at operation.

AORTIC ARCH RESECTION AND GRAFTING FOR ANEURYSM EMPLOYING AN EXTERNAL SHUNT

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THE procedure of choice in the surgical management of aneurysms is excision with restoration of normal blood flow in the parent vessel. The application of this ideal to aneurysms of the thoracic aorta has heretofore been largely restricted to sacciform aneurysms susceptible of local excision and lateral aortorrhaphy.^{1,4} The frequent extension of this principle to the management of aneurysms requiring temporary occlusion of the thoracic aorta to permit safe local excision or resection and grafting has awaited the development of an adequate means of diverting the blood flow during the period of aortic obstruction. The purpose of this report is the presentation of two cases of aneurysm of the thoracic aorta treated by surgical excision and grafting, in which the aortic blood flow was diverted through a shunt of large caliber for periods of six hours and forty-five minutes, and three hours and twenty minutes, respectively.

CASE REPORTS

CASE 1—Fusiform and Saccular Syphilitic Aneurysm of Entire Thoracic Aorta—M J, a 53 year old white man, entered the Albany Hospital on Oct 22, 1953, because of extreme dyspnea. For a number of years the patient had noted a dry cough and exertional dyspnea. During the six month period prior to admission cough and dyspnea had increased. Hoarseness and moderate dysphagia ensued, and a 20 pound weight loss occurred. An abrupt and marked increase in his symptoms occurred two weeks prior to admission, forcing him to stop work as a railroad handy man. His cough became productive of purulent sputum which was increasingly difficult to raise, and cyanosis developed. When admitted to the Albany Hospital, he had required oxygen therapy continuously for thirty six hours. The patient denied chest or back pain. No history of primary syphilis could be obtained.

Physical examination revealed an acutely ill, apprehensive, white man in severe respiratory distress mildly cyanotic despite nasal oxygen, and unable to raise the audible secretions in his tracheobronchial tree because of respiratory obstruction. The temperature was 100° F, pulse 100, respirations 30, and blood pressure 120/70 in each arm. The right lung field was resonant with bronchovesicular breath sounds and a prolongation of the expiratory phase of respiration. The entire left hemithorax was hyperresonant with markedly diminished breath sounds and sibilant rhonchi throughout. The heart was not enlarged, its rhythm was regular, and no murmurs were heard. The remainder of the examination was normal.

Serologic tests for syphilis were positive. The white blood cell count was 21,800 with 96 per cent neutrophil forms. The electrocardiogram revealed a right ventricular hyper

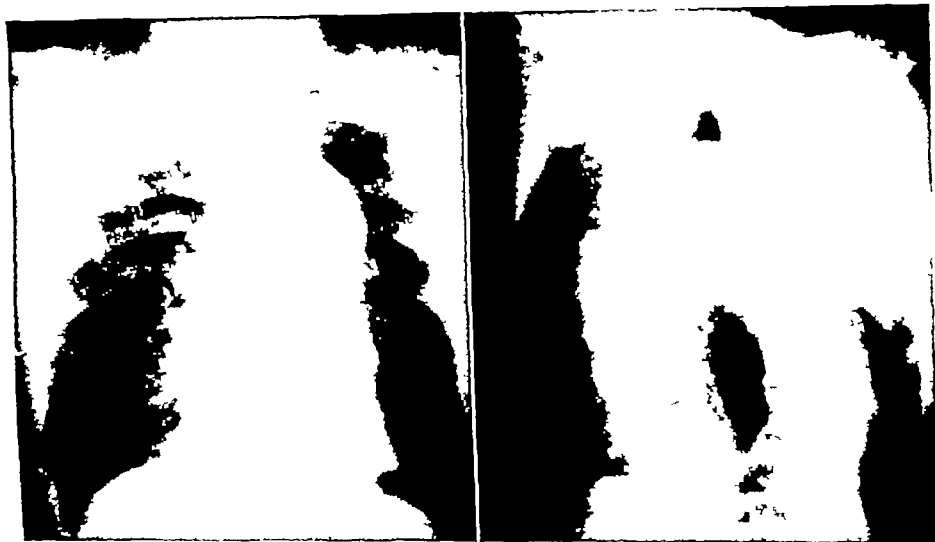
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The opinions and conclusions expressed in this paper are those of the authors and are not to be construed as official or necessarily reflecting those of the Medical Department of the United States Navy, or of the Naval Service at large.

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trophy pattern. Other laboratory tests, including urinalysis, bleeding, clotting, and prothrombin times, nonprotein nitrogen, fasting blood sugar, serum proteins and albumin globulin ratio, and a serum electrolyte pattern were all within the normal range

Roentgenographic and fluoroscopic examinations (Fig 1) revealed a large fusiform aneurysm of the aortic arch and descending thoracic aorta with erosion of the third through seventh dorsal vertebral bodies. There was displacement and compression of the distal



A

B

Fig 1—Case 1. Syphilitic aneurysm of entire aortic arch in a 53-year-old man. A Roentgenogram of chest showing a large fusiform aneurysm of the arch and descending aorta with obstructive emphysema of the left upper lobe and atelectasis of the left lower lobe. The heart is not enlarged. B Oblique projection revealing anterior displacement of the trachea with compression of the left main bronchus. Vertebral erosion was better seen in the right anterior oblique projection.



A

B

Fig 2—Case 1. A retrograde aortogram demonstrates the aortic valve to be competent and delineates the ostia and proximal portions of the coronary arteries which are patent. B A later exposure now shows dye filling the aneurysm and indicates its extent.

trachea and adjacent esophagus. Obstructive emphysema of the left upper lobe and obstructive atelectasis of the left lower lobe were present. A retrograde aortogram delineated the aneurysm more fully. It showed that the aortic valve was competent, and disclosed that the coronary orifices and proximal arterial trunks were widely patent (Fig. 2).

On intensive antibiotic therapy the patient's always improved perceptibly in the ensuing week, and valuable time was thereby afforded to make preparations for a desperate surgical attempt to retrieve an otherwise hopeless situation.

Operation was performed on Oct. 20, 1953, under general anesthesia, with the endotracheal tube advanced to—but not through—the area of tracheal compression. Thoracotomy was performed through the bed of the left 4th rib with transection of the sternum and

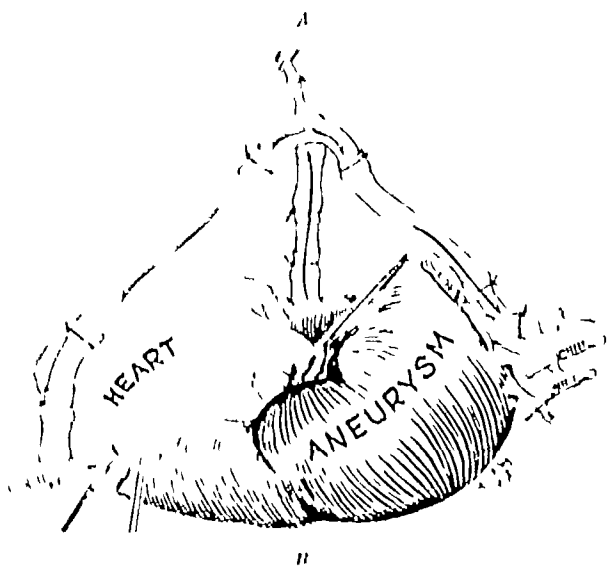


Fig. 1. (A, B). A. Photograph at operation with left lung retracted showing position and size of aneurysm. At the upper right the innominate and left carotid arteries are seen. The left subclavian branch is obscured by the phrenic nerve. The pulmonary outflow tract is visible and intimately attached to the aneurysm. B. Hand-drawn diagrammatic presentation of the operation illustrating the extent of dissection and resection. Inside diameter of aorta 10 mm. (the aorta 4 mm. distal of aortic valve), ascending aorta 15 cm., innominate 9 cm., and descending aorta 4 cm. Clamps have been placed proximal and distal to the aneurysm and on the vessels of the arch with beginning resection of the left lobe. C. View prior to closure of the chest reveals the aortic graft in position and functioning. The aorta has been removed. All anastomoses are complete. The left lung has been removed.

extension of the incision into the right fourth intercostal space. The aneurysm arose in the ascending aorta and involved the entire arch and descending aorta to the diaphragm (Fig 3, A). The aortic window was completely obstructed by the aneurysm through compression of the left main bronchus, which resulted in obstructive emphysema of the upper lobe and atelectasis with suppurative pneumonia of the left lower lobe. Subsequent dissection disclosed compression of the main pulmonary artery, partial erosion of the compressed distal trachea and the adjacent esophagus, and deep erosion of the third to seventh dorsal vertebrae.

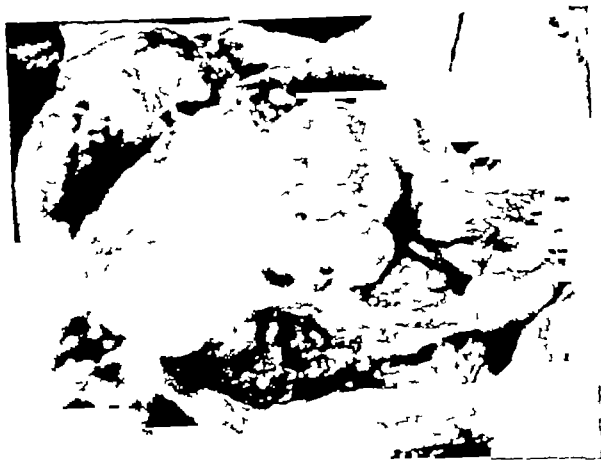


Fig 3 C (For legend see opposite page)

The proximal aorta, great vessels, and distal descending aorta were freed by dissection to permit attachment of a previously prepared three lumbd shunt to by pass the area to be resected (Fig 3, B). The shunt consisted of 1 cm bore Tygon tubing with siliconed glass joints fastened to the ascending and descending aorta by means of heterologous reconstituted freeze dried arterial sleeves. The latter were anastomosed to the host vessel end to side by employing a Beck Pott's exclusion clamp. The innominate artery was cannulated with the third lumb of the shunt, thereby permitting circulation to the head as well as to the trunk during the period of aortic occlusion. The shunt was then filled with saline solution through the air vent at the top to prevent air embolism and placed in operation. The ascending and descending aorta, left subclavian and common carotid arteries were then occluded, and with the shunt operating, the dissection of the aneurysm itself commenced.

In the course of dissection of the region of the aortic window, an opening was made in the attenuated wall of the left pulmonary artery. This necessitated sacrifice of the left lung, which was already useless because of erosive destruction of the left main bronchus. Except for a proximal and distal cuff, the entire thoracic aorta was then removed. The defect was bridged by two reconstituted heterologous (calf) freeze dried aortas⁵ spliced together. The innominate and left common carotid arteries were anastomosed to the arch thus created end to side. The left subclavian artery was intentionally sacrificed. The new arch was then distended with saline solution to remove all air and the clamps removed from the distal aorta, proximal aorta, and left common carotid artery, in the order mentioned. Distended with blood, the graft was obviously too long and had a tendency to kink. This was corrected by making a sleeve resection of 5 cm at the level of the previous splice (Fig 3, C) and again employing the shunt during a forty minute period of occlusion. The reconstructed arch now had an excellent curvature and all suture lines had ceased to ooze. The shunt was therefore removed and the two aortic openings isolated by the exclusion clamp and sutured. The innominate artery was repaired by temporary occlusion

employing two Potts' ductus forceps. During the period of innominate occlusion, circulation to the brain depended upon the left common carotid artery. The chest was then closed with drainage of both pleural cavities connected to a series of water seal and valve bottles that permitted regulation of the intrapleural pressure at mean minus 10 cm. of water pressure.

The operating time was fifteen hours. A blood loss of 5 liters occurred, but was replaced as lost. The shunt was in operation and the aortic arch excluded for six hours and forty five minutes. Because the right brachial artery thrombosed after the preoperative aortogram, no record of the patient's blood pressure was available after the left subclavian artery was sacrificed during the operative procedure. The right axillary and temporal pulses, however, remained full with a regular rhythm throughout the operation. The pulse rate ranged between 80 and 120 per minute. Frequent electrocardiographic tracings revealed no signs of heart strain or coronary insufficiency at any time during the operation. At the conclusion of the operation, there were good bilateral carotid and femoral pulses.



Fig 4—Case 1. Autopsy specimen of heart and entire thoracic and abdominal aorta, with longitudinal incision of aorta and left ventricle. Arrow markers from heart peripherally indicate aortic valve suture closure of proximal shunt limb, proximal anastomosis between ascending aorta and graft, innominate and left carotid anastomoses to graft, graft to graft anastomosis, graft and distal thoracic aortic anastomosis, and the closed incision of the distal shunt sleeve aperture.

Following extubation, the patient responded and opened his eyes when addressed. His color was good. He began to thrash around with his left arm and leg, but the right arm and leg were limp. Right intercostal, diaphragmatic and abdominal muscular function were intact. While the patient was being moved from the operating table, his condition suddenly changed. Immediate reopening of the left chest disclosed that the left pulmonary artery ligature had cut through the vessel and a massive exsanguinating hemorrhage had occurred. The patient did not respond to resuscitative measures. Post mortem examination revealed all suture lines associated with the graft to be intact (Fig 4).

Comment—Aortic arch resection and grafting is a feasible procedure. The shunt employed worked well. The chief objection to the method is the time required to carry out the elaborate procedure. We feel that by improved planning the operating time may be reduced one-third. In the case presented seven major vascular anastomoses and three vascular suture lines to repair the defects left following removal of the shunt were required.

Except for problems arising from mechanical respiratory obstruction, the patient operated upon was in excellent general physical condition. Significant aortic insufficiency or coronary artery disease were fortunately absent in this patient.

Heterologous freeze-dried grafts were used because human material was not available, and probably would not have been of sufficient size for anastomosis to the dilated proximal and distal aortic cuffs.

Whether the partial right hemiplegia would have been permanent in this patient remains in doubt. It must be assumed to have resulted from ischemia caused by prolonged interruption of the left carotid and vertebral arteries. Although interruption of these vessels on one side probably is usually well tolerated, the occurrence of partial hemiplegia in Kirby and Johnson's case¹⁴ of innominate aneurysm following cross-clamping of this vessel for fifty-seven minutes suggests that the maneuver is not uniformly safe. We advocate, therefore, in the future the addition of a fourth limb to the shunt for left common carotid cannulation. This would add but a few minutes to the length of the operation.

CASE 2—Classical Traumatic Aneurysm of the Descending Aorta—T. W., a 25 year old Negro man, sustained a comminuted simple fracture of his right femur, simple fractures of the right second and third metacarpal bones, skull fracture and a mild concussion, together with contusions and abrasions of the chest and abdominal wall in an automobile accident in September 1948, five years before admission. The patient was the driver of the automobile and undoubtedly was decelerated by the steering wheel when the vehicle traveling 55 m.p.h. left the road and struck a tree.

An open reduction of the comminuted fracture of the right femur led to a succession of orthopedic misfortunes which kept the patient in bed or immobilized by plaster spica until September, 1953, when the final cast was removed. A routine photoroentgen film at this time revealed a mediastinal mass projecting to the left.

The history disclosed that in high school the patient had been told he had a heart murmur and advised to stop playing basketball. Chest films taken during high school were not explained to the patient. Soon after the automobile accident the patient experienced short attacks of lancinating pain across his anterior chest. These were followed by the abrupt onset of severe dyspnea which could be relieved by flexing the trunk. No x-ray examinations were made during that period.

Examination following the photoroentgen discovery of the lesion revealed a husky Negro in robust health but with a distinct limp from shortening of the right leg. The blood pressure was 130/80 in both arms. A Grade 2 systolic precordial murmur with maximal intensity over the pulmonic area but transmitted to the neck, left parascapular region, and epigastrium was heard. There was no diastolic component.

Roentgenographic and fluoroscopic examination revealed a thin calcified halo encircling an area of nonpulsatile density measuring 7 by 5 cm. inseparable from the first portion of the descending aorta (Fig. 5 A and B). In the left anterior oblique position the density appeared to project posteriorly and to deviate the esophagus anteriorly and to the right. A retrograde aortogram proved that the lesion was a thin walled aneurysm

arising from the first portion of the descending aorta (Fig 5, C). The contrast material remained in the aneurysm for several seconds following complete clearance of the dye from the aorta itself. No patent ductus was shown in the study.

Bronchoscopy revealed indentation of the membranous wall of the left main bronchus and conversion of its lumen into a crescent shape of approximately 70 per cent normal caliber. The electrocardiogram was normal. Serologic tests for syphilis were negative.

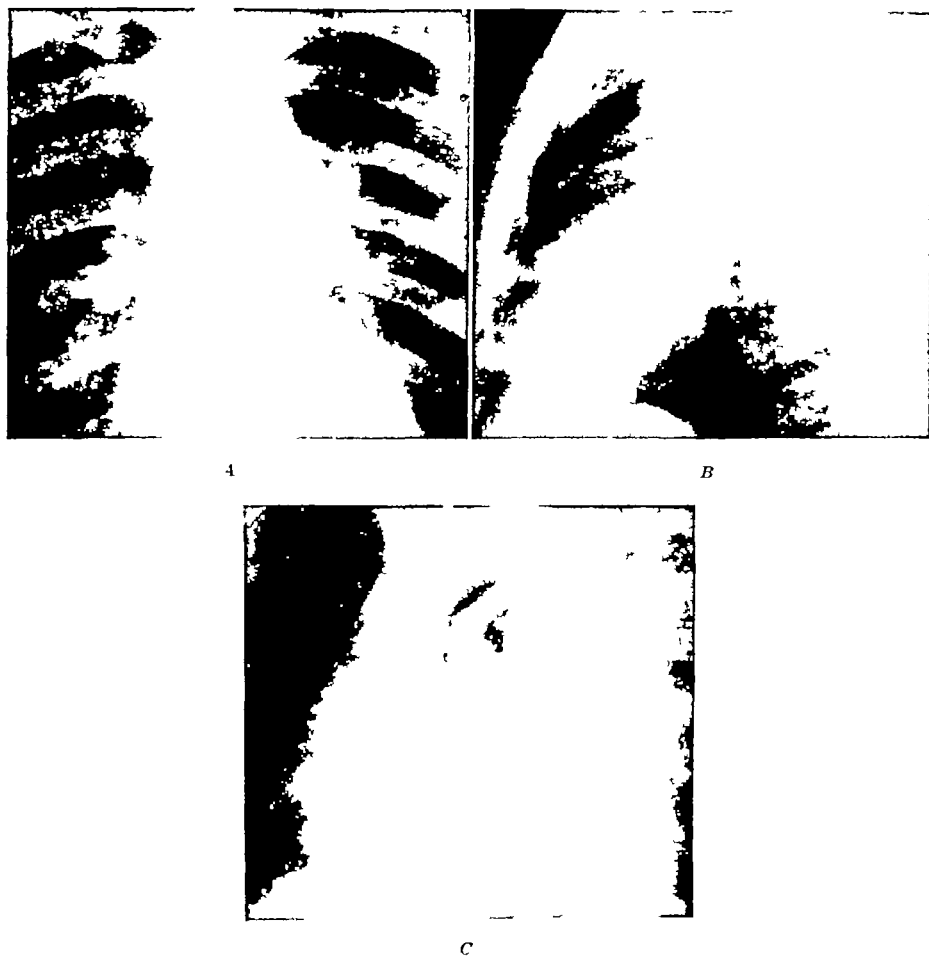


Fig 5—Case 2. Posttraumatic aneurysm of the descending aorta in a 25 year old man. A. Roentgenogram of chest reveals a 7 by 5 cm. density in the left hilum rimmed by a thin layer of calcium. B. On lateral view the lesion is seen at the origin of the descending aorta. C. A retrograde aortogram with dye filling the aortic arch and the aneurysm. Dye remained in the aneurysm for several seconds after the aorta itself was clear of dye.

Operation was performed Dec 3, 1953, under general endotracheal anesthesia. The thoracotomy was done through the bed of the resected fourth rib with posterior fracture of the third, fifth, and sixth ribs to obtain adequate exposure. The aneurysm arose just below the left subclavian artery and encircled three quarters of the circumference of the aorta, to which it was inseparably fused. The dome of the aneurysm was everywhere calcified and very thin walled. There were no pleural adhesions, but a dense subpleural periaortitis with fusion of the aneurysm to the adjacent pulmonary artery, bronchus, and esophagus rendered the dissection somewhat tedious. A patent ductus of small caliber was ligated and divided.

It became evident that the aneurysm could not be tangentially excised, and that it would be necessary to obstruct the flow in the aorta to permit endo aneurysmorrhaphy or resection and grafting. Accordingly, a left subclavian descending aorta shunt, with an inside diameter of 10 mm abruptly tapered to 6 mm to fit the subclavian artery, was introduced in the manner depicted in Fig 6, *A*. With the shunt in operation, the aorta was obstructed above and below the aneurysm and the aneurysm opened. It measured 7 by 5 by 4 cm and an opening from the aorta measured 2.5 to 3 cm.

The technique of endo aneurysmorrhaphy as employed by Bahnson³ was not feasible. Accordingly, the area was resected and the defect bridged by an 8 cm length of homologous freeze dried aorta* obtained from a 19 year old white man, a victim of an automobile accident, shortly after death Aug 8, 1953. The period of aortic occlusion was three hours and twenty minutes. The effect of the shunt on blood pressure proximal and distal to the area of aortic obstruction was recorded by a Hathaway Electronic Recorder† and is shown transcribed in Fig 6, *B*. The operating time was eleven hours. Measured blood loss was 3,450 cc with a replacement of 4 liters.

There was no postoperative evidence of spinal cord damage. The early postoperative period was marred by the development of oliguria and hypotension related to dehydration and low salt syndrome which responded promptly to adequate parenteral therapy. Because of recurrent atelectasis of the left lung, on the third postoperative day a temporary tracheotomy was instituted. The patient's subsequent convalescence was uneventful, and he was discharged from the hospital on the twenty seventh postoperative day. A film of the chest and right brachial and right femoral pulse pressure curves taken five months postoperatively are shown in Fig 7, *A* and *B*.

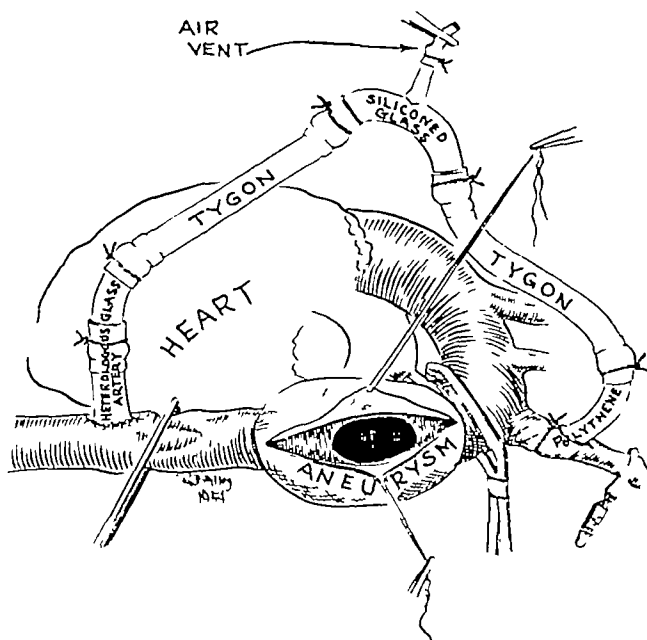
Comment—Although this patient was free of symptoms referable to his aneurysm, it is known that these lesions carry a poor prognosis.¹² It is conjectured that the long period of immobilization imposed by his orthopedic problems may have played a role in his preoperative survival. We may speculate as to whether the small patent ductus played a significant hemodynamic role in relation to lateral intra aortic pressures affecting the aneurysm.

The preoperative aortogram gave a true picture of the relatively small size of the opening in the aorta compared to the aneurysm itself. This information, however, was misleading in the final analysis, because the aneurysm was not susceptible of tangential excision or endo-aneurysmorrhaphy. Without the availability of a shunt and suitable graft, an attempted excision of this lesion doubtless would have led to cross clamping of the aorta and to the attendant risk of permanent neurologic damage.

DISCUSSION

Although in the process of resection of aortic aneurysms the descending aorta has been obstructed for periods up to forty-seven minutes without the production of heart failure or of neurologic damage, experimental and clinical evidence indicates that this maneuver is unsafe except in the management of aneurysms complicating coarctation with its attendant expanded collateral circulation.^{3, 12} The introduction of a shunt to permit diversion of blood flow around an occluded area adds a morbidity factor of its own. In many instances, however, this will be more than compensated for by the time permitted for careful unhurried dissection and excision of a difficult lesion.

*Supplied by the Tissue Bank, United States Naval Medical School, Bethesda, Md.
†Hathaway Instrument Company, Denver, Colo.



A

PROXIMAL TO
ANEURYSM -
AORTA PATENT

BP 104/80
MEAN 94

PROXIMAL TO SHUNT -
AORTA OCCLUDED

BP 180/86
MEAN 126

DISTAL TO SHUNT -
AORTA OCCLUDED

BP 86/84
MEAN 82

DISTAL TO GRAFT -
AORTA PATENT

BP 124/100
MEAN 108

B

Fig 6—Case 2. A Diagram of operation demonstrating position of aneurysm. The aneurysm has been incised and the opening between the aorta and aneurysm depicted. Aorta occluded above and below the lesion with external shunt in place. Also represented is the method of securing the glass joint of the shunt to a sleeve of heterologous freeze-dried vessel (calf) sutured to the aorta. The inside diameter of the shunt was 10 mm except for the polythene subclavian cannula which tapered to a minimum of 6 mm at its flanged end. Over all length of the shunt was 55 cm. B Tracings of records obtained at operation with a 25 gauge needle and recorded by a Hathaway oscillograph. With shunt in operation and the aorta clamped, pressures proximal to the shunt increased. Distally the pulse pressure was virtually absent but an adequate mean pressure was maintained. At completion of the operation with the graft in place and the aorta open, normal pressures were registered distal to the graft.

The risk of hindquarter paralysis resulting from interruption of the descending aorta arises from four factors (1) the level of obstruction (less risk progressing downward from left subclavian artery to abdominal aorta), (2) the duration of obstruction, (3) the number of intercostal artery and other collaterals interrupted in the course of mobilizing the lesion to be excised,

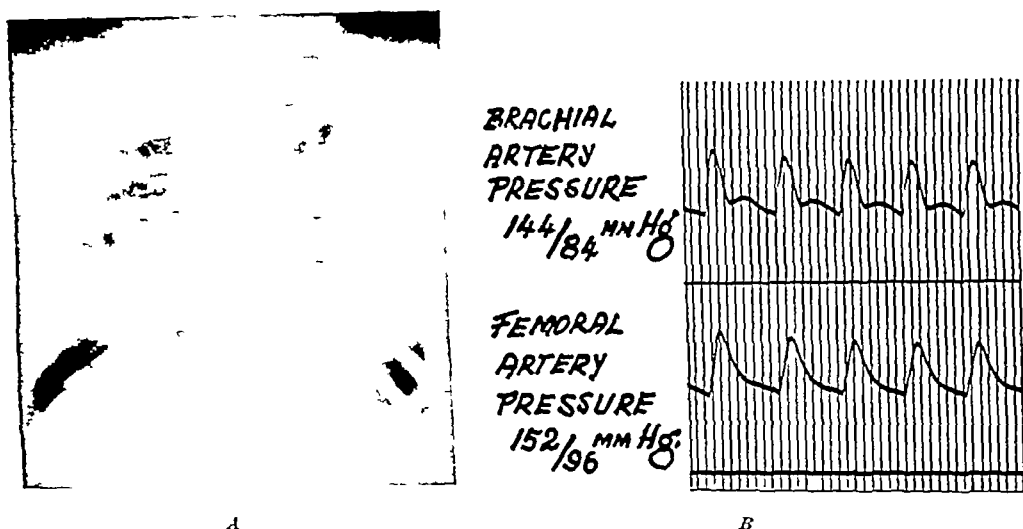


Fig 7—Case 2-4 Chest X-ray four and one half months after operation B Right femoral and brachial arterial pressures obtained at the same time by intra-arterial puncture and recorded by a Hathaway oscillograph

and (4) the metabolic rate of the tissues below the level of obstruction. The combined effects of high aortic occlusion and interruption of all intercostal arteries were doubtless responsible for the partial paraplegia in Lam and Arm's case¹² following aortic occlusion on two occasions of twenty-four and fifteen minutes respectively. Conversely the absence of cord damage following obstruction of the descending aorta for forty-five minutes in DeBakey and Cooley's case¹³ may be related to the low level (eighth dorsal) of aortic obstruction in their case.

Hufnagel and Gross,⁹ by refrigeration of the backs of dogs, and Beattie and his co-workers¹¹ employing hypothermia in dogs, prevented hindquarter paralysis following occlusion of the midthoracic aorta for thirty and ninety minutes, respectively. Hypothermia alone, or in combination with shunts of smaller caliber than those necessary at normal rates of metabolism may be found useful in resectional surgery of the human thoracic aorta. Until measures to minimize the risk of ventricular fibrillation or cardiac standstill in adults under hypothermia are found¹⁰ however, an external shunt of large caliber would appear preferable for cases requiring prolonged occlusion of the thoracic aorta.

The problem of aortic arch resection employing multiple small-bore (1.14 to 2.69 mm) polythene shunts has been studied experimentally by Hardin and associates¹ and these authors report a technically achieved but unsuccessful clinical case of aortic arch resection. They recognized that the small caliber

of the shunts they employed imposed an overwhelming work load on the heart, and contributed to a prohibitive mortality. This was overcome by impeding the inflow of blood into the heart by means of partial occlusion of the inferior vena cava. The maneuver would seem only to add to the complexity of the problem. A simpler solution seems to be the enlargement of the caliber of the shunt.

In selecting the caliber of the shunt to be employed, the closer it approximates the lumen of the vessel to be bypassed the fewer physiologic alterations will result. Izant, Hubay, and Holden¹⁸ have shown experimentally in dogs that a shunt with a 5 mm inside diameter will preserve 80 per cent of the blood flow and adequate pressure to prevent ischemic changes in the spinal cord and kidneys.¹⁹ We have found in dogs that the use of a comparable shunt (6 mm inside diameter) is associated with significant increases in pressure proximal to the shunt which drop to negligible levels if a 10 mm inside diameter shunt is used.²⁰

The optimum size of the shunt for clinical use is not known. The results of the pressure studies in Case 2 and studies on dogs indicates that approximately 10 mm should be the minimum inside diameter. In older subjects with varying degrees of coronary artery disease and hypertension, the increase in cardiac work imposed by shunts of smaller caliber would not be well tolerated.

SUMMARY AND CONCLUSIONS

Two cases of aneurysm of the intrathoracic aorta treated by surgical excision and graft replacement, employing an external shunt of large caliber during the period of aortic dissection and occlusion are presented.

The first case was a fusiform and saccular syphilitic aneurysm of the entire intrathoracic aorta which was excised with restoration of the arch by freeze-dried aortic heterograft. The procedure was technically achieved but the patient expired during recovery from anesthesia as a result of hemorrhage from the pulmonary artery stump of an incidental pneumonectomy. A shunt with a 10 mm inside diameter functioned well during a six hour and forty-five minute period of aortic obstruction.

The second case is a classical posttraumatic saccular aneurysm of the first portion of the descending aorta accompanied by a patent ductus arteriosus successfully treated by partial aortic resection, restoration of aortic continuity by freeze-dried aortic homograft and division of the ductus. Aortic pressures taken during the operation of the shunt disclosed distal aortic pressures to be adequate for prevention of renal and spinal cord ischemia. It is estimated that an external shunt for the adult human aorta should have a minimum inside diameter of 10 mm throughout.

The false preoperative estimate that the second case would be amenable to local excision and anastomophy suggests that a shunt and suitable graft should always be available when a case of intrathoracic aneurysm is explored.

The shunt employed in the cases presented not only provides a conduit during the period of aortic obstruction but introduces a factor of safety while the aneurysm is being dissected.

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(For Discussion see page 102)

RESECTION OF THE THORACIC AORTA WITH REPLACEMENT BY HOMOGRAFT FOR ANEURYSMS AND CONSTRICTIVE LESIONS

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ONE of the most significant advances in the field of cardiovascular surgery made during the past decade has been the development of the concept and technique of excisional therapy for certain congenital and acquired lesions of the aorta. The first successful resection of a segment of the aorta was reported by Gross³¹ and Crafoord and Nylin¹⁵ in 1945, both of whom excised a coarctation of the aorta and performed end-to-end anastomosis. In lesions where this can be accomplished, this procedure still provides the most effective method of therapy. On the other hand, in certain unusual types of coarctation, a lengthy segment of aorta must be removed thus precluding the performance of end to end anastomosis. Various methods of dealing with such situations have been proposed but these also impose certain limitations upon operability. In some cases of atypical coarctation for example the problem may be solved by utilizing the left subclavian artery as a shunt to bridge the aortic defect by a method used experimentally by Blalock and Park.⁸ In general, however, the problem of restoration of aortic function after extensive aortic resection required the development of a technique with wider applicability in order that more extensive resections could be satisfactorily accomplished. Aortic resection for lesions such as aneurysms not associated with vascular obstruction also imposed certain limiting factors upon the operation, control of which largely determines the success of aortectomy.

At the turn of the century Carrel¹² demonstrated experimentally that successful resection of the thoracic aorta in animals was possible and that the resulting defects in the aorta could be successfully repaired by vessel grafts. During the past decade the feasibility of aortic homografts used clinically to repair extensive surgical defects in the aorta created by resection of coarctation has been demonstrated by Gross³² and subsequently by others^{3, 9, 10, 21, 24, 27, 29, 39, 41, 46} who have utilized the procedure in the treatment of aneurysms and constrictive lesions located in both the thoracic and abdominal aorta. The present limitations on the applicability of aortic resection include certain technical and physiological factors among which are the nature and extent of the lesion and its location. The purpose of this presentation is to consider these factors and the means of combating their limiting influence in a series of 12 aortic

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resections and homograft replacements, 7 of which were done for aneurysms and 5 for constrictive lesions (See Addendum) In some cases grafts preserved in a modified Tyrode's solution were employed for this purpose, but in the majority of instances freeze-dried or lyophilized grafts were used The criteria of selection and method of procurement, preservation and final restoration of the graft for use are reported elsewhere²⁰

ANEURYSMS

Excisional therapy of thoracic aortic aneurysms was suggested and tried unsuccessfully by Tuffier¹¹ more than fifty years ago In 1910 Cannel¹² stated "The diagnosis of the aneurisms of the aorta can be made today at a comparatively early period It is rational to think that some of them can be extirpated Therefore we must find methods which permit repair to the aortic wall after a partial or complete resection" Yet not until two years ago was extirpation of aortic aneurysms first advocated as the procedure of choice in all cases of thoracic aneurysm where factors permitted its satisfactory application¹⁴ The inevitably fatal nature of aortic aneurysms justifies an aggressive approach to such lesions directed toward removal of the irreparably damaged tissue and restoration of aortic continuity

In general two types of procedures may be employed for this purpose, namely tangential excision with lateral aortorrhaphy and resection of the involved segment of aorta with replacement by aortic homograft For sacciform lesions involving the ascending aorta and proximal portion of the aortic arch the former procedure may be used By placing a clamp across the neck of a sacciform aneurysm in the proximal regions of the aorta the sac distal to the clamp may be excised and the aorta repaired without encroachment upon its lumen This is obviously an important practical method of dealing with sacciform lesions in the ascending aorta and arch since it precludes the necessity of temporary arrest of the circulation which at a high aortic level could easily produce lethal ischemic damage to the brain as well as left ventricular dilatation and fatal cardiac failure

Fusiform aneurysms of the thoracic aorta which involve the entire circumference of the vessel present a special problem in that extirpation requires removal of an entire segment of the aorta Aneurysmectomy for such lesions presents two problems, one is concerned with the ischemic effect on tissues distal to the point of aortic occlusion and the other with bridging the defect created by aortic resection The latter appears to have been satisfactorily solved for the present by the use of preserved aortic homografts Thus solution of the former problem represents the critical factor in successful performance of the operative procedure A number of factors may influence the degree of ischemic damage occurring during the period of temporary interruption of aortic circulation The level of occlusion is obviously significant Even brief periods of occlusion of the ascending aorta or the transverse arch of the aorta are likely to be fatal Thus for the present lesions in this region remain inoperable by clinically proved methods On the other hand temporary occlusion of the thoracic aorta distal to the left carotid or left subclavian artery may be tolerated by the myocardium if certain undesirable sequelae from

ischemia to the tissues distal to the occlusion can be controlled satisfactorily. Experience indicates that the spinal cord is the most vulnerable tissue to temporary aortic occlusion at these high levels. The degree of renal ischemia

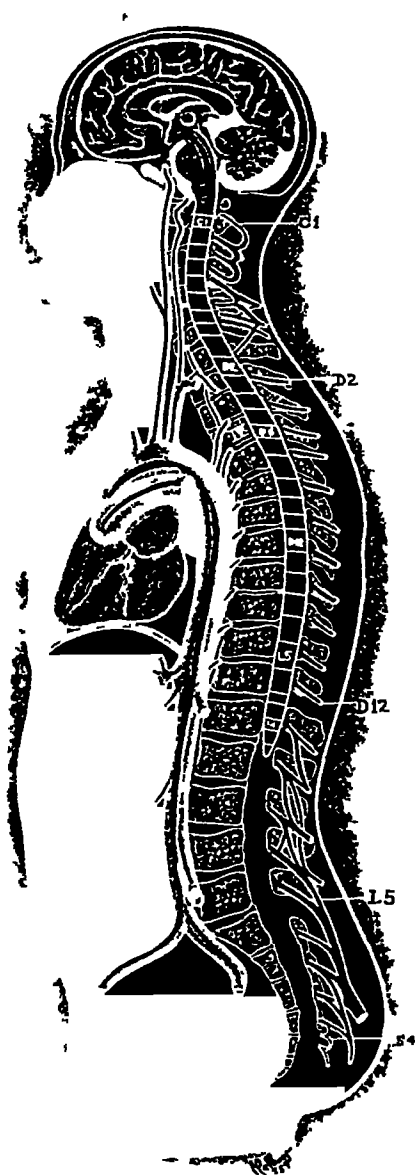


Fig. 1.—Diagrammatic drawing of sagittal section of body showing relationship between the anatomic levels of aorta, vertebral column and spinal cord.

produced by temporary aortic occlusion above the renal arteries has not resulted in significant impairment of renal function in our experience, nor has

there been any evidence of ischemic damage to other abdominal viscera or the lower extremities. Thus prevention of spinal cord damage during occlusion of the descending thoracic aorta is usually the critical factor in the success of the procedure.

In general the level and the duration of occlusion of the aorta determine the extent of cord damage. The distal portion of the aortic arch is located approximately at the level of the fourth thoracic vertebra and the origin of the sixth dorsal nerve from the spinal cord and the aortic hiatus in the diaphragm is approximately at the level of the fifth lumbar nerve (Fig 1). The lower end of the spinal cord is located opposite the first or second lumbar vertebra. Whereas cross-clamping the thoracic aorta distal to the arch may produce a severe paraplegia or even death from spinal shock, occlusion of the abdominal aorta below the renal arteries as in resection of arteriosclerotic aneurysms in this region rarely is complicated by spinal cord damage. Thus in 45 cases of abdominal aneurysms in which the aorta distal to the renal arteries was occluded for periods up to two hours there has not been a single instance of neurologic change in the lower extremities after operation.

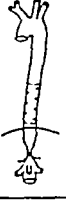
Another factor influencing the degree of ischemic damage to the spinal cord is the necessity for double-clamping the aorta above and below the lesion in order to excise the intervening segment. Experimental evidence indicates that cross clamping at two sites simultaneously, one on the proximal and the other on the distal thoracic aorta, increases the paraplegia and mortality rates significantly in animals.⁴² Furthermore, in aneurysmectomy the risk is increased by the necessity for division of varying numbers of intercostal arteries depending upon the extent of the lesion. In the experimental animal division of intercostal arteries concomitantly with temporary aortic cross-clamping increases the incidence of paraplegia as well as the mortality. Thus it would appear that the length of the lesion influences significantly the risk of excisional therapy since this necessitates division of increasing numbers of intercostal arteries.

Still another important consideration is the extent to which collateral circulation develops around the lesion. In instances of insidious or chronic occlusion of the aorta as in coarctation, the rich collateral network which is usually present insures against serious ischemic damage occurring even during prolonged periods of aortic cross-clamping. Consequently few cases of paraplegia resulting from resection of the aorta for coarctation have been reported.^{5, 7, 10} Conceivably collateral circulation may be mildly stimulated by the presence of an aortic aneurysm and tissues below the lesion may even develop a relative tolerance to chronic partial ischemia. Although theoretically these factors may exert some protective influence, their clinical effectiveness in preventing ischemic changes during temporary aortic occlusion cannot be relied upon.

Still another factor influencing the degree of ischemic damage produced by arrest of aortic circulation is the duration of occlusion. The critical safe period of aortic occlusion has not been accurately determined, being quite

variable in the reported experience. Bahnson² reported cross clamping the distal portion of the aortic arch for 7 minutes in one case and 20 minutes in another without evidence of ischemic cord damage, and Crafoord¹⁹ reported, in a case of patent ductus arteriosus, occluding the aorta for 27 minutes without paralysis. Our experience indicates that the safe period may extend even

TABLE I A RESECTIONS OF THE THORACIC AORTA WITH REPLACEMENT BY HOMOCRAFT FOR ANEURYSMS

Case	Blood Pressure	Lesion	Etiology	Date of Operation	Method of Repair	Result	
						Functional	Blood Pressure
8							
EL 35yr W♂	LA 260/160 LL 0/0		Acquired Arteriosclerotic	6-16-53		Fair	LA 160/110 LL 110/90
EL 35yr W♂	LA 160/110 LL 110/90		Same 2nd operation	10-26-53		Excellent	LA 140/90 RL 170/100
9							
SS 12yr W♂	RA 155/90 LA 110/90 LL 0/0		Congenital	8-17-53		Excellent	RA 124/60 LA 100/60 LL 130/70
10							
AT 20yr W♂	RA 190/110 RL 0/0		Congenital	9-15-53		Excellent	RA 140/90 RL 170/100
11							
JH 30yr W♂	RA 190/110 LA 130/140 RL 0/0		Congenital	10-27-53		Excellent	RA 150/90 LA 120/90 RL 130/100
12							
JOG 29yr W♀	RA 160/90 LA 160/110 RL 0/0		Congenital	3-26-54		Excellent	RA 150/90 LA 146/90 RL 150/110

longer (Table IA) Nevertheless the duration of occlusion in operations for thoracic aortic aneurysms must be minimized regardless of the factors which may tend to prolong this critical safe period

Accordingly certain technical steps in the operation designed to minimize the period of aortic occlusion assume practical importance In general the method which we have used in all cases has been similar to that employed in Case 2 which will be used as an illustrative example (Figs 2-3) Adequate exposure is achieved by posterolateral thoracotomy, one usually resects a rib for this purpose Where a long segment of aorta must be visualized, more satisfactory exposure is obtained by dividing the posterior ends of several adjacent ribs In an occasional instance as in Case 6 in which the entire thoracic aorta is involved it may be practical to make a double entry into the pleural space by opening both the fourth and the eighth intercostal spaces in order to expose adequately the proximal and distal ends of the lesion (Fig 4) All preliminary dissection and mobilization of the adjoining aorta are completed and temporary encircling tapes are applied After resectability of the lesion is determined the graft is prepared for use²⁰

With everything in readiness the proximal and distal clamps are applied and the exact time of occlusion is noted Then the aneurysm is quickly removed larger bleeders being ligated as they are divided In extensive lesions total removal of the aneurysm at this moment would involve a tedious dissection with possible damage to adjacent organs thus delaying the ultimate restoration of aortic circulation Under such circumstances only enough of the aneurysm is removed initially to permit trimming of the proximal and distal ends of a normal aorta and to provide a trough for placement of the graft (Fig 3) Complete evacuation of the aneurysm and excision of the sac are postponed until after the graft has been inserted and aortic flow has been resumed In performing the anastomosis considerable time may be saved by utilizing a simple over-and-over continuous suture using 0000 arterial silk interrupting the suture line at two or three points along the circumference of the anastomosis Upon opening the clamps there may be temporary bleeding from needle holes but this will cease usually after a few minutes Occasionally additional sutures may be required in the anastomosis When aortic continuity is completely restored the remainder of the sac is removed If erosion of the vertebral bodies has occurred there is ordinarily no sac present posteriorly and the laminated thrombus is in contact with the marrow cavity of vertebrae In some instances where the graft is suspended across such a large excavation, it is advisable to wrap the graft either with some adjacent pleura or else to transplant into the thorax a pedicled flap of omentum which may be brought through the aortic hiatus in the diaphragm Aortic occlusion time in the chest should not exceed one hour and in less extensive lesions may be approximately thirty minutes Anticoagulants are not used during or after such operative procedures

In addition to minimizing the duration of aortic occlusion other possible solutions to this problem include the use of shunts around the occluded seg-

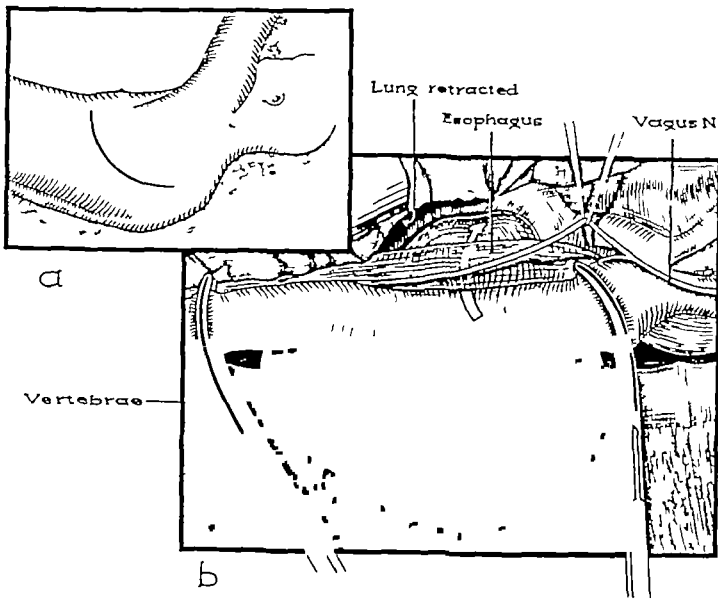


Fig 2—Drawing showing operative technique employed in Case 2 used as illustrative example of procedure usually employed in resection of descending thoracic aorta for aneurysm. *a* Left posterolateral thoracotomy incision is employed dividing posterior ends of adjacent ribs. *b* Aneurysm is demonstrated in relationship to vertebrae posteriorly and esophagus and lung anteriorly. Arrow indicates retroesophageal projection of aneurysm to the right.

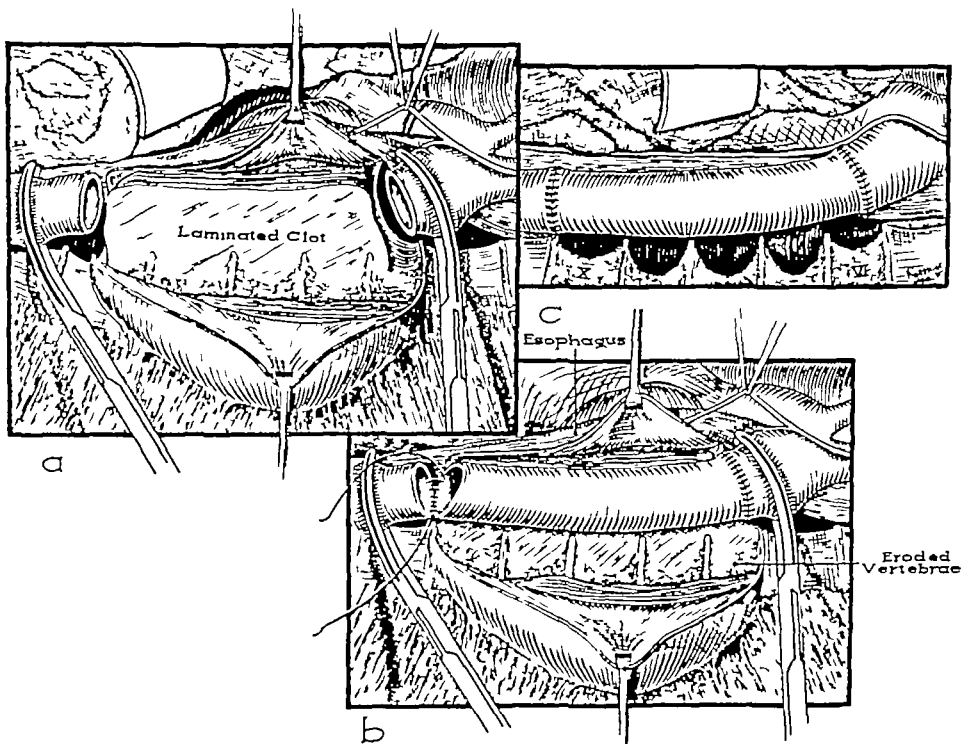


Fig 3—Drawing showing method of insertion of aortic homograft to restore circulatory continuity. *a* After clamping aorta above and below lesion a portion of the aneurysm is excised and the ends of the aorta trimmed. *b* Craft lies in the lumen of the incompletely excised aneurysm as the anastomoses are performed with continuous over and over suture of 0000 art rial silk interrupted at several points. Upon restoration of circulatory continuity of aorta remainder of aneurysm and contents are removed. *c* Craft which bridges vertebral excavation is covered with adjacent pleura.

ment of aorta and the use of hypothermia. The former may be achieved by temporary intraluminal or external shunts.^{13 16 21 37 38 43} Both of these methods have been employed experimentally and although they would appear to be quite useful for this purpose their clinical effectiveness has not been demonstrated. Another possible solution to this problem lies in the use of hypothermia in order to reduce the arterial oxygen requirements of the central nervous system and thus prolong the period of safe aortic occlusion.⁶ The protective effect of hypothermia in preventing ischemic damage during experimental temporary occlusion of the aorta has been clearly demonstrated.^{4 42}

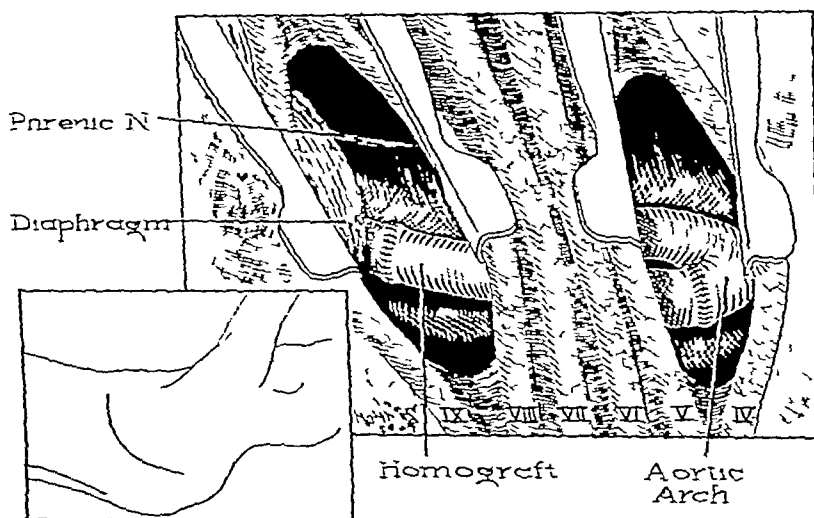


Fig. 4.—Drawing showing incision used in Case 6 where length of the lesion required exposure of entire descending aorta. Single incision is made through skin and soft tissues and pleural cavity is opened through both the fourth and eighth intercostal spaces facilitating performance of the aneurysmectomy and anastomoses.

In our own experiments with temporary aortic occlusion the mortality in 50 control animals was 32 per cent and in a similar number of hypothermic animals 25 per cent. In these same experiments the rate of paraplegia in the surviving controls was 65 per cent and in the hypothermic group no instance of paraplegia was encountered. From these observations it may be concluded that hypothermia deserves cautious clinical trial particularly with operations for the more proximally situated aneurysms of the descending thoracic aorta. Local cooling of only the spinal cord proper by local application of cold packs to the spinal column may reduce both the temperature of the cerebrospinal fluid and the oxygen requirements in the spinal cord. An advantage of the latter technique is that some of the systemic factors may be eliminated which in the general hypothermia somewhat dangerous particularly when used in patients of advanced years, but further experience will be necessary before spinal cooling can be recommended for this purpose. We have used general hypothermia twice (Cases 5 and 6) and local or spinal hypothermia once (Case 7).

and although the thoracic aorta was occluded for 53, 62, and 53 minutes, respectively, in none of these was there evidence of paraplegia after operation.

The following case reports of our experience with resection of aneurysms of the thoracic aorta emphasize certain anatomic and physiologic considerations encountered in the excisional therapy of aortic aneurysms and represent a consecutive series of 7 patients treated by aneurysmectomy and aortic homograft.

CASE REPORTS (ANEURYSMS)

CASE 1—**R. A.** a 46 year old white man, was admitted to Methodist Hospital on Dec. 31, 1952, with excruciating pain of three months' duration located in the lumbar region of the back, abdomen and inguinal region, requiring opiates for relief. Palpation of the left costovertebral angle elicited pain, and there was roentgenographic evidence of an aneurysm of the lower thoracic and upper abdominal aorta producing erosion of the bodies of the ninth, tenth, eleventh, and twelfth thoracic and the first lumbar vertebrae. Serologic test for syphilis was positive.

On Jan. 3, 1953 through a left thoracoabdominal incision, the aneurysm was resected, and the 15 cm. defect in the aorta was repaired with a homograft preserved in modified Tyrode solution (Table IA). The period of aortic occlusion was 45 minutes, and there were no neurologic sequelae after operation. Convalescence was uneventful and he was discharged from the hospital thirty one days later.

The patient has remained well and free of pain and has returned to work. Recent roentgenograms of the thoracolumbar spine reveal progressive regeneration of the eroded vertebral bodies and there is no evidence of dilatation of the graft.

Comment—To our knowledge this is the first successful resection of an aneurysm of the thoracic aorta with homografting. Lam and Aram³⁹ previously reported a similar case in which a large part of the aneurysmal sac and contained thrombus were not removed, resulting in infection several months later with fatal secondary hemorrhage. It is important, as emphasized previously^{2, 11, 12, 39} to extirpate as much of the aneurysm and thrombus as possible.

The homograft which lay suspended across the large vertebral excavation in our Case 1 lacked soft tissue support so this was supplied by utilizing the pedicled omentum to wrap the graft. Follow up period in this case is approximately sixteen months, and the graft shows no evidence of degeneration.

CASE 2—**O. T.** a 50 year old Negro man, was admitted to Jefferson Davis Hospital, June 3, 1953 with excruciating pain in the left chest requiring opiates for relief. In 1942 he had experienced substernal pain radiating to the back, and in 1947 he was admitted to the hospital because of severe pain in the left chest following the course of the fifth and sixth intercostal nerves. On that admission roentgenogram of the chest revealed an aortic aneurysm and destruction of the thoracic vertebral bodies. Serologic tests for syphilis were positive and the patient received 6 million units of penicillin. Prior to the present admission he had been observed on several occasions in the medical clinic for the aneurysm and during this period he was almost totally disabled by pain in the back and left chest.

Physical examination revealed a thin, chronically ill appearing man in constant discomfort. There were normal carotid and brachial pulsations and blood pressure was 140/90 mm. Hg. The heart was mildly enlarged and there was no evidence of aortic insufficiency. There was dullness to percussion over the posterior lung fields, and breath sounds were absent in this area. There was a palpable pulsation in the posterior chest wall near the

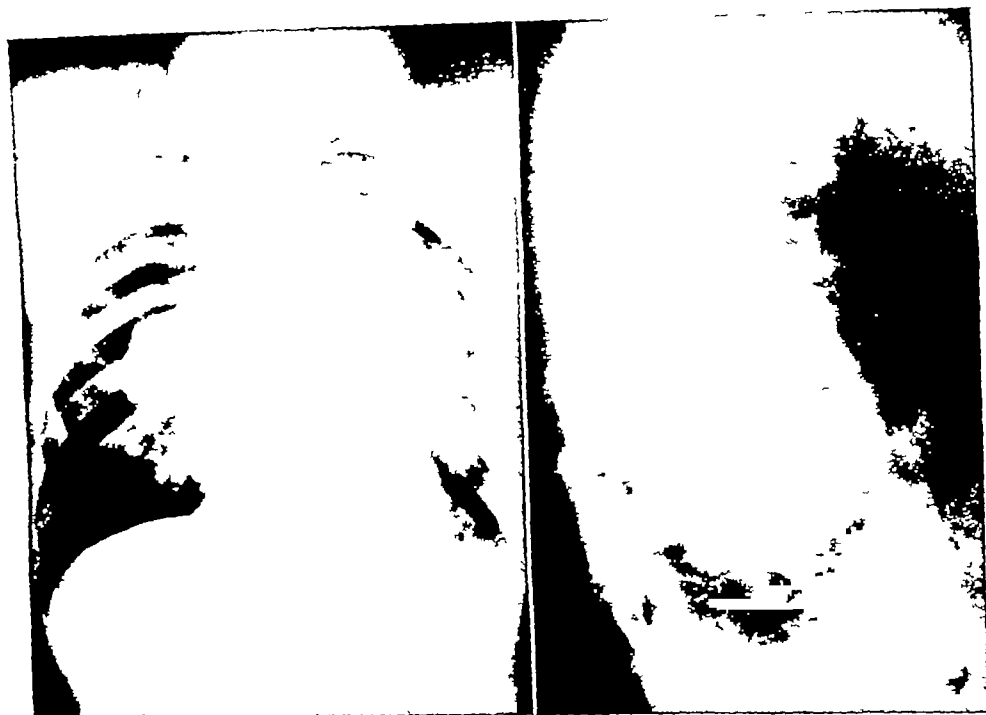


Fig 5—Roentgenograms of chest in Case 2 demonstrating in the posteroanterior projection (A) an extensive aneurysm of the descending thoracic aorta presenting both to left and right of mediastinal shadow and in the lateral (B) the retroesophageal position of lesion displacing esophagus and trachea forward.

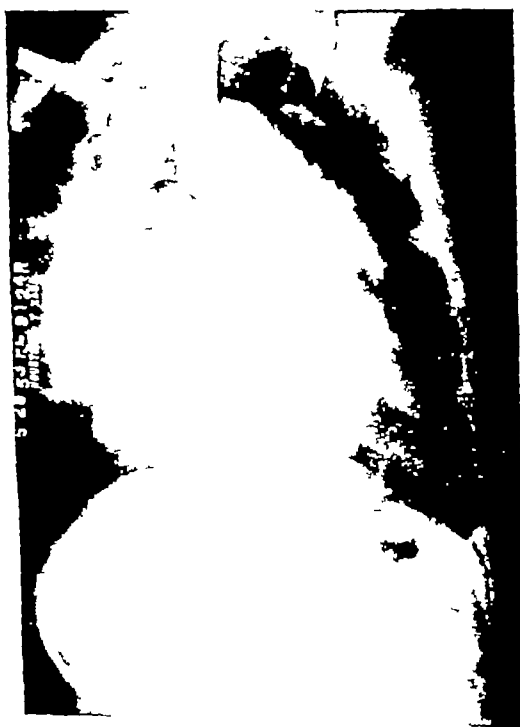


Fig 6—Oblique roentgenogram of the chest in Case 2 showing striking distortion of esophagus and extensive erosion of bodies of sixth through tenth thoracic vertebrae.

inferior angle of the left scapula. The remainder of the examination was not remarkable. Hemogram and urinalysis were within normal limits, and serologic tests for syphilis were negative.

Roentgenograms of the chest revealed a large mass in the posterior mediastinum extending to the left with another process extending to the right (Fig 5). Esophagrams showed a distortion of the esophagus with displacement to the left (Fig 6). There was erosion of the bodies of the sixth through the tenth thoracic vertebrae. Fluoroscopy revealed minimal pulsation in the mass.

On June 11, 1953, a left thoracotomy was performed through the bed of the resected seventh rib, and to obtain adequate exposure the posterior ends of the fifth, sixth, and eighth ribs were divided. The aneurysm was extensive and was densely adherent to the posterior surfaces of the left upper and lower lobes of the lung. Proximally, the aneurysm arose about 15 cm distal to the left subclavian artery and extended distally to a point about 5 cm above the aortic hiatus in the diaphragm (Table IA). Posteriorly, there was no definite sac, and there was a deep excavation of the vertebral bodies leaving the intervertebral bodies relatively uninvolved. There was an extension of the sac far to the right of the midline and posterior to the heart, and this was encroaching upon the ribs in the right costovertebral gutter. The aneurysm, together with the contained laminated thrombus, was completely removed, and a 23 cm homograft preserved in modified Tyrode solution was inserted (Figs 2 and 3). The graft preserved in modified Tyrode solution was inserted (Figs 2 and 3). The period of aortic occlusion was 45 minutes. At the completion of the procedure circulation to the lower half of the body was satisfactory. A prophylactic tracheostomy was performed.

Urinary output during the first twenty-four hours after operation was 750 cc, and no renal complications ensued. His general condition improved steadily, and after about ten days he was completely free of pain and his appetite returned. On the second day postoperatively it was noted that there was diminished sensation in the feet and general weakness of all muscles of the lower extremities. This weakness never developed into frank paralysis, and he had no loss of sphincter control of the anus or bladder. After intensive physical therapy he left the hospital ambulatory on Aug 17, 1953, using a cane for support.

Since dismissal from the hospital, the patient has improved progressively, and when last examined on April 15, 1954, he had gained 30 pounds and walked unassisted with minimal residual weakness in his legs. Roentgenograms of the chest at this time showed progressive resolution of the residual pleural thickening on the left. There was kyphoscoliosis in the region of the vertebral erosion, but callus formation indicated satisfactory healing (Figs 7 and 8).

Comment—In spite of the magnitude of the lesion in this case, successful surgical correction was achieved, and almost the entire descending thoracic aorta was replaced by a homograft measuring 23 cm in length. The neurologic sequelae, although troublesome, were not disabling, and the patient has been completely rehabilitated. The result in terms of palliation is striking, and life expectancy has unquestionably been extended.

CASE 3—T. M., a 58-year-old man, was admitted to Jefferson Davis Hospital on Nov 10, 1953, complaining of intermittent, severe pain in the left lower chest posteriorly radiating to the epigastrium. He was in good health until he sustained rib fractures on the right in an automobile accident in June, 1953.

He was in constant discomfort and appeared chronically ill. Blood pressure was 138/90 in both arms, and the heart was slightly enlarged with a Grade II aortic systolic murmur. There was dullness to percussion over the left chest posteriorly, but no abnormal pulsations.

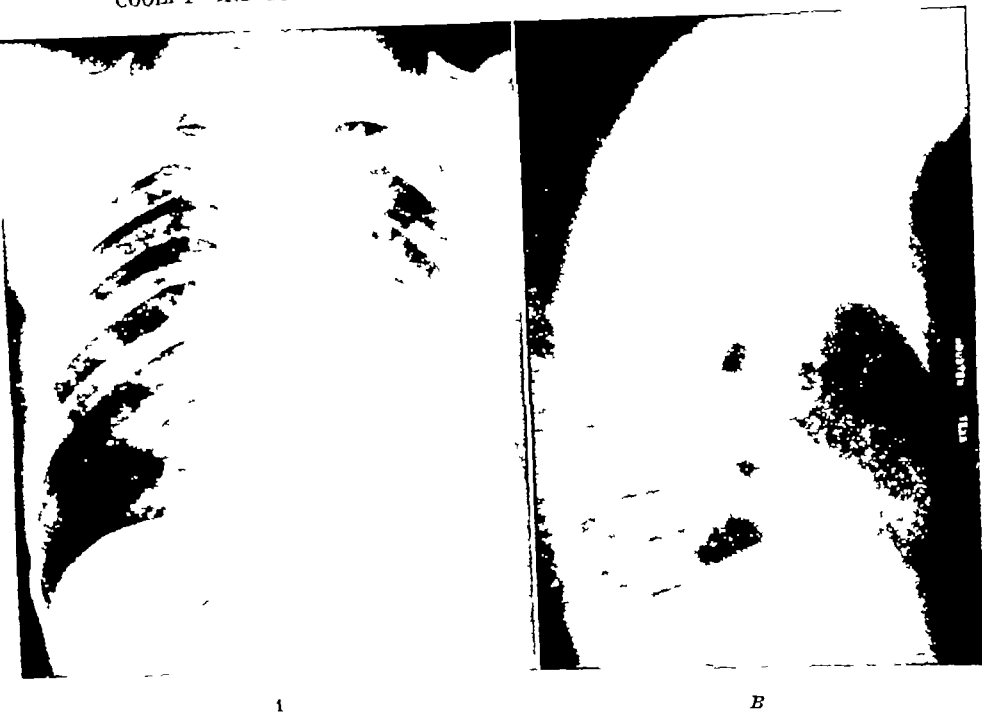


Fig 7—A and B Roentgenograms of the chest in posteroanterior and lateral projections in Case 2 made ten months after operation, demonstrating absence of aneurysm and residual pleural thickening on left.



Fig 8—Oblique roentgenogram of thoracic vertebrae in Case 2 made ten months after operation showing moderate kyphoscoliosis and progressive healing with callus formation in the involved vertebrae.

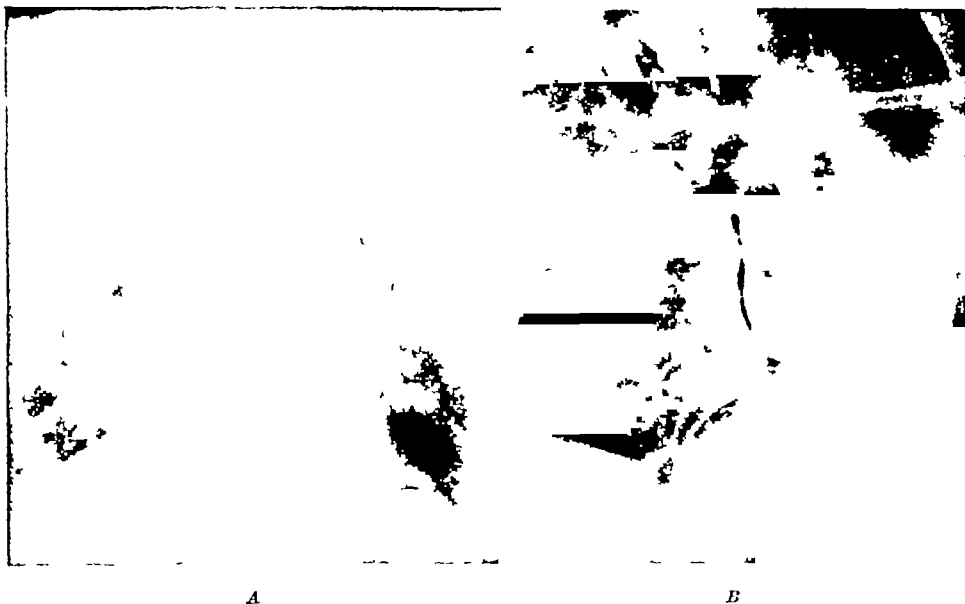


Fig 9—Roentgenograms of chest in Case 3 showing in posteroanterior projection (A) a tortuous fusiform aneurysm of the entire descending thoracic aorta and in the oblique (B) deviation of the esophagus by aortic elongation and kinking



Fig 10—Petouched retrograde aortogram in Case 3 demonstrating elongated aorta and fusiform aneurysm with distal fusocylindrical dilatation containing laminated thrombus

Serologic test for syphilis was positive. Electrocardiogram revealed left ventricular hypertrophy and suggested anterolateral ischemia.

Roentgenograms of the chest showed moderate dilatation of the ascending aorta and an extensive aneurysm involving the entire descending thoracic aorta (Fig 9). These findings were confirmed by a retrograde aortogram (Fig 10).

He received 2,400,000 units of penicillin daily for five days prior to surgery. Following retrograde aortography he was hypotensive for twenty-four hours, probably due to impaired myocardial function.

Thoracotomy was performed Nov 9, 1953, under ether anesthesia. A single posterolateral incision was made through soft tissues but in order to expose the aorta proximal and distal to the aneurysm the pleural cavity was entered in both fourth and eighth inter spaces (Fig 4). The fusiform aneurysm extended from the level of the left subclavian

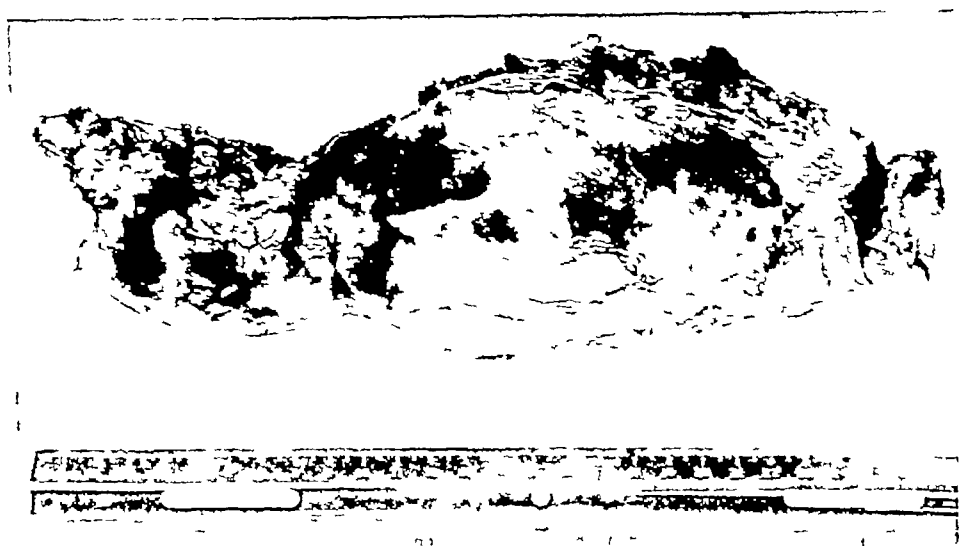


Fig 11—Photograph of specimen removed in Case 3 showing aneurysm involving entire descending thoracic aorta.

artery proximally to the level of the aortic hiatus distally (Table IA). The upper portion of the aneurysm was not as large as the distal portion where the lesion was approximately 12 cm in diameter and was eroding the vertebrae (Fig 11). The aorta was clamped above and below the aneurysm for 77 minutes while a segment of aorta measuring 30 cm in length was resected and replaced by a lyophilized homograft. Operation lasted 4 hours and 45 minutes and he received a 3,500 cc blood transfusion.

During the postoperative period a constant drip of Levophed (nor-epinephrine) was necessary to maintain blood pressure at $\approx 100/50-60$ mm Hg. He moved his lower extremities well and urinary excretion was satisfactory. About twelve hours after operation Levophed was inadvertently discontinued and before it could be restarted the patient became restless, suddenly hypotensive and expired.

Autopsy revealed that the aortic homograft was intact and presumably functioning satisfactorily. The heart showed no evidence of recent infarction although there was coronary arterio-sclerosis and histologic evidence of myocardial fibrosis.

Comment—In this case the aortic homograft measuring 30 cm in length was the longest used in our series. The entire descending aorta was removed from just below the left subclavian artery to the aortic hiatus in the diaphragm.

dividing all of the intercostal arteries. In spite of this extensive aortic resection the patient moved his lower extremities well following operation. Adequate exposure was obtained by utilizing the technique of double entry into the chest by separate intercostal incision. The decision to remove the entire aneurysm involving the entire descending thoracic aorta was probably a mistake, and only the distal portion of the aneurysm which was eroding vertebrae and causing severe pain should have been removed. Obviously, removal of the entire lesion increased the operative risk.

CASE 4—E. M., a 50 year old Negro woman, was admitted to Jefferson Davis Hospital on Nov. 12, 1953, with severe pain in the left chest of eighteen months' duration. The



A

B

Fig. 12—A and B. Roentgenograms of the chest in Case 4 in posteroanterior and lateral projections showing fusiform dilatation of the entire thoracic aorta with fusosacciform protuberance in the distal half of the descending aorta.

pain originated posteriorly near the inferior angle of the scapula and radiated obliquely anteriorly and inferiorly. Opium was necessary for relief of pain. She had lost 20 pounds in weight.

Physical examination revealed a poorly developed and chronically ill appearing woman who was in moderate discomfort. The blood pressure was 170/100 mm Hg. The heart was enlarged and there was an aortic systolic murmur Grade II, but no evidence of aortic insufficiency. The remainder of the examination, including a neurologic survey, was not remarkable.

The serologic test for syphilis was positive, but the remainder of the laboratory studies were considered normal.

Roentgenograms of the chest revealed moderate cardiac enlargement, mostly left ventricular (Fig. 12). The entire aortic arch was involved in a fusiform aneurysmal dilata-

tion In the distal half of the descending thoracic aorta, there was a fusosacciform aneurysm occupying the costovertebral sulcus posteriorly but demonstrating no erosion of the vertebrae or ribs

On Nov 19, 1953, under ether anesthesia a left posterolateral thoracotomy was done through the seventh intercostal space There was a large fusiform dilatation of the entire aortic arch and proximal descending aorta (Table IA) The aneurysm in the lower thoracic aorta was fusosacciform and extended over a distance of approximately 10 cm The aortic diameter proximal to the lesion was about twice the size of the distal aorta, and the lesion extended to the diaphragm below (Fig 13) Because of the distribution of the pain, only the aneurysm of the lower thoracic aorta was resected The lesion was



Fig 13—Photograph of specimen removed in Case 4 showing fusosacciform aneurysm with discrepancy in diameter of the aorta proximal and distal to lesion

pressing on the vertebrae and ribs posteriorly, but erosion of bony structures was absent Continuity of the aorta was re established by inserting a 12 cm lyophilized segment of thoracic aorta (Fig 14) When the clamps were removed 38 minutes after they had been applied circulation to the lower half of the body was re established

Her postoperative course was smooth, with no evidence of any neurologic disturbances, and she was discharged from the hospital on Dec 13, 1953 When last seen in



Fig. 14—Photograph made at operation in Case 4 showing 12 cm. homograft inserted into aortic defect. Lower anastomosis is located about 5 cm. above aortic hiatus in diaphragm



A

B

Fig. 15—A and B Posteroanterior and lateral roentgenograms of chest in Case 4 made five months after operation showing persistence of fusiform aneurysm of the aortic arch and absence of fusiform lesion of the descending thoracic aorta

the chest clinic on April 6 1954, she was free of pain and had gained 15 pounds. Roentgenograms of the chest revealed progressive clearing of the pleural thickening at the left base (Fig 15), and fluoroscopy revealed no dilatation in the graft.

Comment—In this case only the symptomatic portion of the aneurysm, i.e. the fusosacciform dilatation in the lower thoracic aorta, was resected. In contrast to Case 3 where the entire descending thoracic aorta was removed and death ensued, this patient has responded well. Apparently the sacciform protruberances on the aorta represent the site of maximum degeneration and weakness and are most likely to cause pain and ultimately to perforate.



Fig 16—A and B Posteroanterior and lateral roentgenograms of chest in Case 5 made six weeks after aneurysmectomy performed elsewhere showing large aneurysm of distal half of aortic arch.

CASE 5—D. A 17 year old white girl was admitted to Methodist Hospital on Dec. 25, 1953 with a diagnosis of aneurysm of the aortic arch and suspected septicemia. She had been healthy and was attending public high school where a routine survey roentgenogram of the chest revealed a small cystic mass in the region of the left upper lobe. Exploratory thoracotomy was done on Nov. 11, 1953, at another hospital, at which time a small thin-walled aneurysm of the arch of the aorta measuring about 2.5 cm. in diameter was discovered arising just distal and adjacent to the ligamentum arteriosum. This lesion was excised tangentially and aortorrhaphy was accomplished with considerable difficulty due to the thinness and friable nature of the neck of the lesion. After this operation she ran a febrile course and septicemia was suspected but blood cultures were sterile. She was discharged from this hospital about three weeks later but readmitted in three weeks again with fever 106° F, and one spell of convulsions. In spite of intensive antimicrobial treatment, fever persisted,

and she developed severe substernal pain and a "brassy" cough. Blood cultures were persistently sterile. Serial roentgenograms of the chest revealed an expanding mass in the upper left chest adjacent to the mediastinum.

She was then seen in consultation and transferred to Methodist Hospital. Roentgenograms of the chest upon admission revealed the mass to measure 8 by 9 cm. in diameter representing a rapid increase in size of the lesion during the previous twenty-four hours (Fig. 16). Laboratory studies revealed a moderate anemia but no other abnormalities.



Fig. 17—Photograph made at operation in Case 5 showing aortic arch homograft inserted to restore aortic as well as left subclavian circulation.

Operation was performed on Dec. 25, 1953, under general anesthesia and hypothermia, which was induced in a special rubberized blanket containing coils through which a refrigerant solution was circulated. When the rectal temperature was reduced to 85° F., the blanket was removed and the operation was begun. Upon opening the previous thoracotomy incision it was apparent that the mass in the mediastinum was a large false aneurysm of the aorta intimately adherent to the left upper lobe (Table IA). The aortic arch was mobilized proximal to the lesion and the mid-thoracic aorta mobilized distally. A proximal clamp was applied across the arch of the aorta distal to the left common carotid artery, and a distal clamp was applied several centimeters below the lesion. Dissection of the aneurysm revealed that it had a false sac supported largely by the left lung. The aneurysm contained mostly fresh clots and originated from the site of the previous aortorrhaphy which had disrupted a portion of left subclavian artery. A portion of left subclavian artery was excised with the aneurysm, and the defect in the arch

was replaced by a corresponding segment of lyophilized homograft of aortic arch measuring 7 cm in length (Fig 17). The patient's subclavian artery was anastomosed to the graft. Aortic occlusion time was 53 minutes and the left subclavian was occluded an additional 12 minutes. The operation required 4 hours and 10 minutes. Rewarming of the patient was accomplished in the rubberized blanket, through which warm solution was circulated, and by use of heat cradles.

Her condition for the first three days was satisfactory. Urinary output was excellent, and there were no neurologic sequelae. However, oral temperatures steadily increased thereafter to a range of 101° to 103° F and tachycardia persisted (130 to 160 beats per minute). At intervals she received penicillin, streptomycin, erythromycin, achromycin, Chloromycetin and polymyxin in large doses without response. On the fifth postoperative day a positive blood culture for *Klebsiella pneumoniae* was finally obtained and this organism was resistant to all antibiotics. Because of an increasing shadow in the chest films demonstrated at the left base several unsuccessful thoracenteses were performed. In spite of repeated blood transfusions she expired on the eighth postoperative day.

Autopsy revealed multiple intrapulmonary abscesses and a hemothorax on the left. No definite source of bleeding was evident and the anastomoses were intact. There was also no evidence of infected vegetations in the graft or on the heart valves.

Comment—General body hypothermia was used in this critically ill patient because the location of the aneurysm made it apparent that cross-clamping of the aortic arch would be required. It is significant, therefore, that in spite of the fact that the aorta was occluded just beyond the level of the left common carotid artery for almost one hour no neurologic damage resulted. Indeed the hypothermia appeared to play a vital part in increasing the patient's tolerance to an extensive operative procedure. It appears that she would have survived operation if the organism causing the septicemia could have been controlled. In this connection and in light of certain observations^{10, 11} suggesting that tissue cooling may enhance infection, the hypothermic procedure in this case may have contributed to the difficulty in controlling the septic process.

CASE 624—W. M., a 31-year-old white man, was admitted to Methodist Hospital on July 5, 1953, with a history of having sustained a severe crushing injury to the chest in March, 1954, while on active duty in the Air Force. Hemothorax resulted which was successfully treated by thoracenteses and since that time he was asymptomatic. Roentgenographic studies on admission revealed a partially calcified mass in the distal aortic arch. At thoracotomy on July 7, 1953, a fusiform aneurysm arising in the arch of the aorta just distal to the left subclavian artery and about 6 cm long was identified. Partial aneurysmectomy was done excising tangentially the lateral projection of the lesion and aortorrhaphy was done with multiple interrupted silk sutures. Postoperative convalescence was uneventful and he was dismissed ten days later.

He remained well until December, 1953, when he complained of pain in the left chest, hoarseness, and difficulty in deep breathing. Roentgenograms of the chest suggested a recurrence of the aneurysm, and aortography confirmed the diagnosis. A second operation was done on Feb. 5, 1954, under general hypothermia. Body temperature was reduced gradually to 32° F. The aneurysm had recurred and enlarged so that it involved the entire aortic arch beginning at a point just distal to the left common carotid artery and extending about 8 cm along the aortic arch and descending thoracic aorta distally (Table I). The aorta was doubly clamped just distal to the left carotid artery and also distal to the aneurysm and the lesion was resected preserving the attachment of the subclavian artery. The 8 cm defect in the aorta was restored with a comparable segment of aortic homograft preserved in modified Tyrode solution. The period of aortic occlusion

was 62 minutes. At the completion of the operation, which lasted 4 hours and 20 minutes, the patient's temperature was 83° F. Rewarming was accomplished with heat cradles and the special blanket circulating hot water.

About six hours after operation a massive hemothorax developed and reexploration and evacuation of the blood clots were necessary. A generalized ooze of blood from all raw surfaces was noted, and intravenous calcium gluconate, vitamin K, and protamine sulfate in addition to blood transfusions were used. The chest was closed with drainage. Recovery was complete, and he was discharged from the hospital fifteen days later. Since that time he has remained well and returned to work.

Comment—To our knowledge, this is the first successful use of hypothermia for thoracic aneurysmectomy (See Addendum). Soon after the second operation there was evidence of a massive left hemothorax which required operative evacuation. There was an apparent temporary disturbance in the clotting mechanism producing a definite bleeding tendency after use of hypothermia. In this instance the bleeding tendency was apparently controlled by injection of calcium, vitamin K, and protamine sulfate, although the exact cause for the disturbance was not ascertained. The possible relationship of the hypothermic state to the clotting mechanism is not entirely clear.

CASE 7—J. B., a 66 year old Negro man, was admitted to Jefferson Davis Hospital Feb. 25, 1954, with severe aching pain in the left chest of six months' duration and a weight loss of 17 pounds. He had a history of a penile lesion fifteen years previously but received no treatment.

Physical examination revealed a blood pressure of 190/120. The trachea was deviated to the right, but there was no tracheal tug. The heart was slightly enlarged to the left, and there was a Grade II aortic systolic murmur and frequent premature beats. The left chest posteriorly was dull to percussion.

Serologic test for syphilis was positive. Electrocardiogram suggested anterolateral ischemia and left ventricular hypertrophy.

Roentgenograms of the chest showed a dilated ascending aorta and large fusosacciform aneurysm of the entire descending thoracic aorta (Fig. 18).

In spite of the fact that the patient was an obviously poor candidate for surgery, because of the disabling symptoms present operation was undertaken on March 4, 1954. Prior to operation local refrigeration of the spine by means of ice packs was employed for about three hours. During this interval the temperature of the spinal fluid decreased from 93.2° to 85° F and body temperature decreased from 98.4° to 94.5° F. He described numbness in the feet and legs, but motor function was normal.

A left thoracotomy was performed resecting the sixth rib. The aneurysm arose about 3 cm. distal to the left subclavian artery and extended to a point 5 cm. proximal to the diaphragm (Table IA). The lung was adherent to the mass, and posteriorly the sac was absent, and the adjacent vertebrae and ribs were eroded. The defect in the aorta which measured about 18 cm. after the lesion was excised was bridged with a lyophilized aortic homograft. The aorta was occluded for 53 minutes. The operation lasted 3 hours and 25 minutes and he received 3,000 cc. blood transfusion. Blood pressure was 100/60, and his condition was fair at the conclusion of the procedure.

During the ensuing two hours he recovered from anesthesia and moved his lower extremities and talked to the attendants. About four hours after operation his blood pressure dropped to 80/50 in spite of intravenous Levophed (nor epinephrine). A roentgenogram of the chest was obtained, and a massive left hemothorax was demonstrated. The patient's condition deteriorated rapidly, and while preparation was being made for operation he suddenly expired.

At autopsy the left chest contained an estimated 2,000 cc of liquid unclotted blood. No definite bleeding point was demonstrated, and the graft was intact. A generalized hemorrhagic ooze was the probable cause of the hemothorax. There was evidence of coronary sclerosis and focal myocardial fibrosis.

Comment—In this elderly patient it was considered too risky to employ general body hypothermia; therefore local application of cold to the spine was used to reduce the temperature of the cerebrospinal fluid. Although death occurred six hours after operation, he responded well enough in the postoperative interval to move his lower extremities voluntarily and appreciate painful stimuli. Whether or not the local hypothermia employed here had any influence on the subsequent bleeding tendency is debatable, but at the second thoracotomy it was evident that there was a capillary type of bleeding which was generalized.

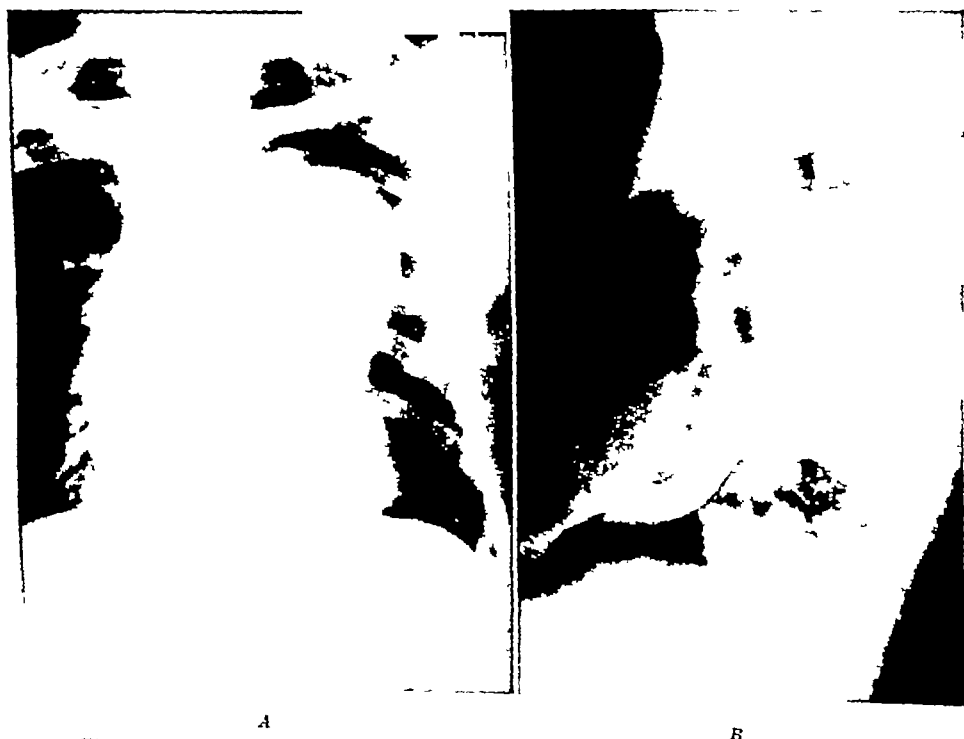


Fig 15—A and B Posteroanterior and lateral roentgenograms of chest in Case 7 showing fusiform dilatation of aortic arch and fusosacciform aneurysm of proximal half of descending thoracic aorta.

CONSTRICTIVE LESIONS

Lesions producing an obstruction to circulation through the descending aorta may be either congenital or acquired. The usual congenital coarctation occurs in the isthmic region of the aortic arch where it joins the descending thoracic aorta just distal to the origin of the left subclavian artery and the ductus Botalli. In the typical coarctation only a short segment of aorta is

obstructed and local resection with end-to-end anastomosis is relatively simple and the procedure of choice. In these cases blood pressure in the carotid and subclavian arteries is elevated, but in the lower half of the body it is diminished and may be unobtainable. In the so-called "infantile" coarctation, the aortic constriction lies in a more proximal position, often above the left subclavian artery and usually involving a longer segment of aorta. Although in such cases death usually occurs shortly after birth, some patients survive and may live to adulthood. In those instances where the coarctation lies proximal to the left subclavian artery there may be a striking discrepancy in the pulse and blood pressure obtainable in the two arms and hypertension in the right arm may be extremely high. The length of the constrictive segment in many of these cases together with its position makes resection with end-to-end anastomosis a virtual impossibility. Thus, in these unusual types of coarctation the procedure of aortic resection with restoration of circulation necessitates the use of aortic homografts in order to bridge the resulting defect. We are reporting two such cases (Cases 9 and 11) in which restoration of circulation through the aorta as well as through the left subclavian artery was achieved by employing a corresponding segment of aortic arch homograft.

In a significant number of cases aneurysms occur in the vicinity of the coarctation arising either from the aorta itself or from the point of origin of the dilated intercostal and bronchial arteries. In order to remove the pathologic constriction along with the secondary aneurysms, homografts should be available to bridge the defect.

Acquired constrictive lesions of the thoracic aorta are rare. In general they tend to occur farther along the course of the aorta than the usual site for congenital coarctations. There appears to be a localized aortitis resulting in segmental atherosclerosis producing the obliterative process in the aorta. The length and position of the process varies, but in the majority of cases which have been reported the involved segment measures approximately 4 to 6 cm. in length and is located in the middle or terminal thirds of the descending thoracic aorta (Table II). Only twelve such lesions have been reported, and in only six has surgical exploration been done. In the three cases in which surgical excision and placement of an aortic homograft were performed, the results were excellent. We are reporting a fourth case of this type in which there was a coarctation of the distal thoracic aorta and also a separate constriction in the upper abdominal aorta causing obliteration of the celiac axis (Case 8).

The operative procedure of resection with replacement by aortic homograft essentially as done for aneurysms of the descending thoracic aorta, may also be employed for constrictive lesions. A favorable feature of the constrictive lesions in contrast to aneurysms is the presence of a well developed collateral network of vessels which provide adequate circulation to the tissues distal to the point of temporary cross-clamping and for almost indefinite periods of time. Thus, in these cases there is no need for haste in completing the resection and grafting after the clamps are applied. Furthermore there

is no necessity for using shunts or hypothermia to protect distal tissues during periods of temporary occlusion. Emphasis should be placed on complete restoration of all major vascular channels, particularly the left subclavian artery when its origin is involved in the obliterative process. The following cases of aortic resection and replacement by homograft for constrictive lesions of the aorta are presented to illustrate some of the technical features of this problem.

TABLE II REPORTED CASES OF COMPRESSION OF THE THORACIC AORTA AT UNUSUAL SITES

AUTHOR	AGE (YR.)	SEX	LOCATION	SURGICAL TREATMENT	RESULT IN OPERATIVE CASES
1 Schlesinger (1935) ⁴⁵	15	F	Distal third	None	
2 Hasler (1911) ²²	40	M	Distal third	None	
3 Costa (1930) ¹	40	F	Middle third	None	
4 Hickl (1931) ⁴⁶	38	F	Middle third	None	
5 Hahn (1933) ³⁴	44	M	Distal third	None	
6 Schleickat (1933) ⁴⁴					
6 Olm (1949) ⁴⁴	20	F	Distal third	Aortic resection without repair	Died 5 hours later
7 Bahnson et al (1949) ¹	17	F	Middle third	Dorsal sympathectomy	Slightly improved
8 Freeman et al (1950) ²⁸	14	F	Distal third	None	
9 Beattie et al (1951) ²	19	F	Distal third	Aortic resection homograft	Excellent
10 Glenn et al (1952) ²⁰	19	F	Distal third and upper abdominal	Bypass using splenic artery	Good
11 Brock-Graham (1952) ¹⁸	28	M	Distal third aneurysm located proximal to stenosis	Aortic resection homograft	Excellent
12 Deterling (1952) ²⁷	23	F	Distal third obliterated left subclavian artery	Aortic resection homograft	Excellent
13 Cooley-DeBakey (1954) (Case 8)	33	M	Distal third proximal abdominal	Aortic resection homograft	Excellent

CASE REPORTS (CONSTRUCTIVE LESIONS)

CASE 8—E. L., a 35-year-old white man, was admitted to Methodist Hospital on June 14, 1953, with a history of hypertension known to be present since 1939. He was rejected for military service on account of hypertension but had continued to work as a boiler maker. On April 7, 1953, while at work he was seized with a sudden headache and lost consciousness. Systolic blood pressure at that time was more than 300 mm. Hg. He developed a complete left hemiplegia which gradually improved and at the time of admission he had slurring of speech and left hemiparesis. Because of religious beliefs he refused to receive a blood transfusion in any form.

Physical examination revealed an underdeveloped, chronically ill appearing man who was apparently emotionally unstable. Blood pressure in the left arm was 260/160 mm. Hg and left leg 0/0. The heart was not enlarged and there were no murmurs, although a was accentuated. Moderate development of collateral vessels was present in the chest wall and a systolic bruit was audible over the mid-thoracic spine to the left of the midline.

Röntgenogram of the chest revealed erosion of the inferior aspect of the right ninth rib. Scattered areas of calcification were present anterior to the lower thoracic and upper

TABLE IB RESECTIONS OF THE THORACIC AORTA WITH REPLACEMENT BY HOMOGRAFT FOR CONSTRUCTIVE LESIONS

Case	Lesion	Etiology	Date of Operation	Method of Repair	Length of Graft	Period of Occlusion	Result
1 R A 46yr W♂		Syphilitic	1-5-53		15 cm.	45 min	Excellent
2 O J 50yr N♂		Syphilitic	6-11-53		23 cm	45 min	Excellent Slight weakness in legs
3 T M 58yr W♂		Syphilitic	11-9-53		30 cm	77 min	Died 12 hours later Shock
4 E McC 50yr N♀		Syphilitic	11-19-53		12 cm	38 min	Excellent
5 D J 17yr W♀		Congenital (Recurrent)	12-25-53		7 cm	53 min hypotherm	No neurologic sequelae Septicemia Died 8 days later
6 W McC 31yr W♂		Traumatic (Recurrent)	2-5454		8 cm	62 min hypotherm	Excellent No neurologic sequelae
7 J B 66yr N♂		Syphilitic	3-4-54		18 cm	53 min spinal-hypotherm	Died 6 hours later Shock

lumbar vertebral bodies, suggesting calcification of the aorta. The arch of the aorta was normal in appearance and the site of coarctation was not visualized. An aortogram done in another hospital was unsatisfactory.

On June 16, 1953, a left posterolateral thoracotomy was done entering the chest through the bed of the resected fifth rib. The collateral vessels in the chest wall were not as well developed as would be expected in the usual coarctation. The arch and proximal descending aorta were normal. Beginning in the lower third of the descending thoracic aorta there was a constriction and almost complete occlusion of the aorta ex-

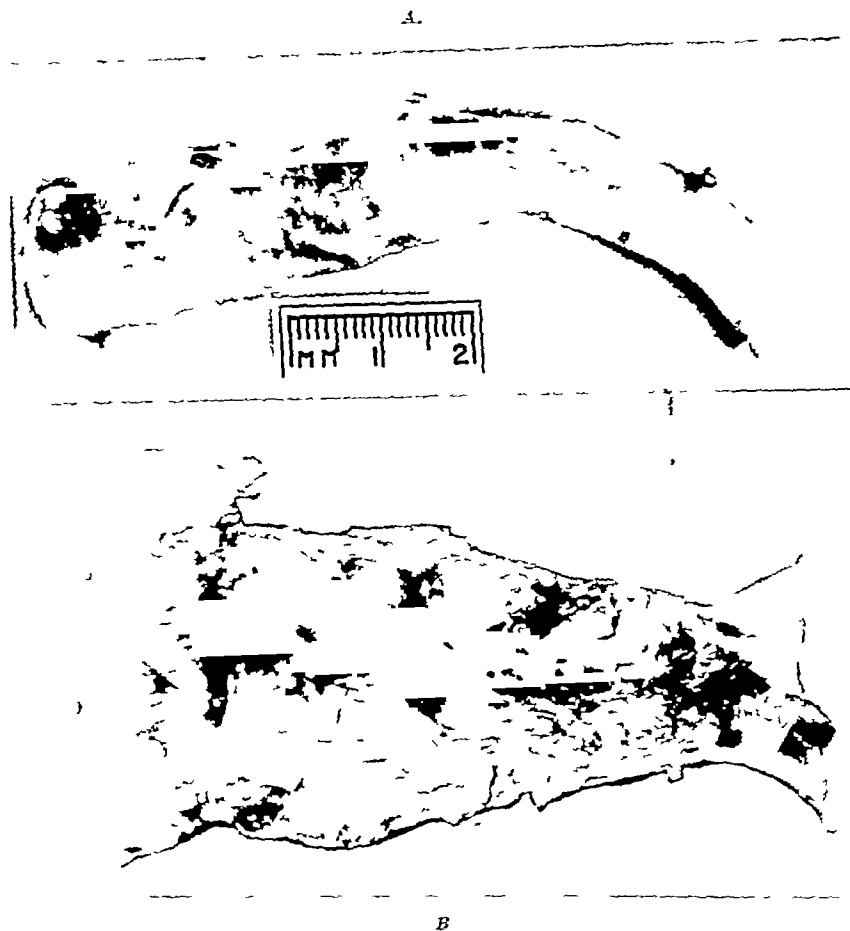


Fig. 19.—Photograph of specimen in Case 8 showing (A) the irregular cone-shaped configuration of the constricted segment, and, in opened specimen (B) the ragged atherosclerotic lumen with almost complete obstruction at the lower end.

tending over a distance of approximately 8 cm. (Table IB). The constriction was surrounded by a zone of fibrosis, scarring and multiple small collateral vessels. The sclerotic process appeared to end at the aortic hiatus where the aorta was again soft and of normal texture. In order to gain proper access to the lesion in the lower aorta it was necessary to resect a segment of the eighth rib and enter the pleural cavity at that level. The constricted segment of aorta was then excised between clamps and the resulting defect was bridged with a segment of aortic homograft preserved in modified Tyrode's solution measuring 9 cm. in length.

His recovery was complicated by emotional instability and mental confusion. Upon discharge from hospital on June 28, 1953, there were pulses in the femoral arteries which

were palpable but not very forceful. Blood pressure in the arms was 180/110 mm Hg and in the legs was 160/120.

The specimen measured about 9 cm in length and contained heavily calcified atherosclerotic plaques (Fig 19). Near the distal end the orifice was about 3 mm in diameter. The tissue at the margins of the specimen appeared normal in texture and the arteriosclerotic process ended abruptly. During the ensuing months which he spent at home, the emotional instability and mental confusion referable to the previous stroke improved somewhat. On Oct 20, 1953 he was readmitted to the hospital. Blood pressure in the left arm was 160/110 and in the left leg 110/90. Pulses in the legs were weak.



A

Fig 20—A and P. Aortogram and explanatory diagram in Case 8 made four months after first operation showing graft in place and second constriction involving the proximal abdominal aorta.

Retrograde aortography revealed a constriction in the proximal portion of the abdominal aorta just above the superior mesenteric and renal arteries and occluding the celiac axis (Fig 20). The previously inserted thoracic homograft revealed no evidence of dilatation.

On Oct 26, 1953 a left posterolateral thoracotomy was done through the ninth inter space. The diaphragm was detached posteriorly and the left crus of the diaphragm and the aortic hiatus were opened. In the proximal abdominal aorta there was an occlusion similar to the one previously resected in the terminal thoracic aorta (Table IB). The abdominal occlusion which was arterio-sclerotic and calcified, measured about 2.5 cm in length and obliterated the origin of the celiac axis which was represented by a fibrous cord (Table IB).

The previous graft was exposed and its terminal portion was mobilized and excised along with the segment containing the constriction. A lyophilized homograft, approximately 5 cm in length was inserted to bridge the defect between the graft in the thoracic aorta proximally and the abdominal aorta distally (Fig 21). The graft, which had been in place for four months was in good condition.

His postoperative convalescence was smoother than after the first operation, and he was discharged from the hospital in good condition on Oct 26, 1953.

When last examined on Feb 15 1954, blood pressure in the right arm was 140/90 mm Hg, right leg 170/100. Pulses in the lower extremities were full and bounding and he was entirely free of headaches.

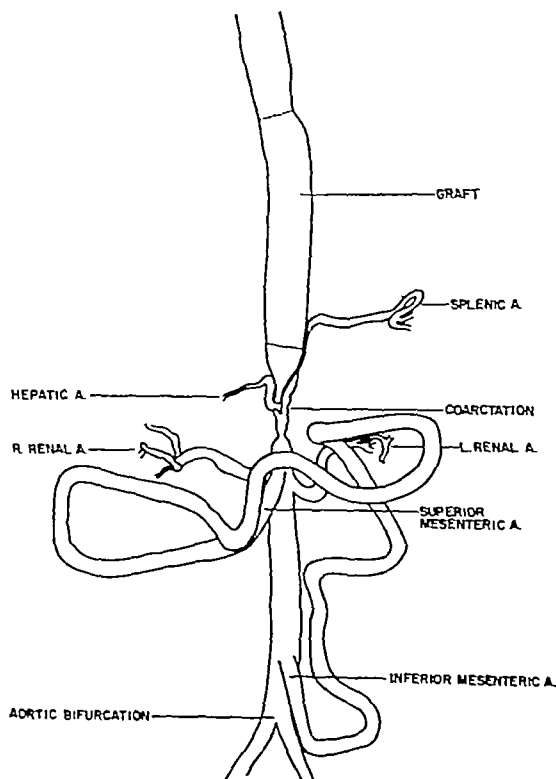


FIG 20 B (For legend see opposite page)

Comment—This case resembles others which have been previously reported in which the thoracic aorta in young patients is obliterated by an arteriosclerotic process confined to a localized segment of aorta usually in the middle and distal thirds. It is unique in that there was a short 'skip area' at the aortic hiatus in the diaphragm where the aorta was uninvolved, although there was another constriction involving the proximal abdominal aorta. The celiac axis was chronically obliterated by this latter process and gradual occlusion of this vessel had produced no ill effects.

The second operation provided an opportunity for examination of the thoracic graft which had been inserted four months previously. After the

terminal end of the graft was mobilized, it was occluded and the lower end was amputated for histologic study and will be reported elsewhere.²⁰ Grossly the graft was supple and elastic, and the intimal surface was smooth and glistening. Anastomosis to the lower end of the graft was satisfactory, and the sutures had no tendency to tear out. Because of the patient's religious convictions, no blood transfusions were given during either operation, and blood substitutes were unnecessary.

CASE 9—S S, a white boy, aged 12 years, was admitted to Methodist Hospital on Aug 13, 1954, with mild cramps in his legs usually occurring at night. Several months prior to admission, while swimming, he developed severe precordial pain lasting several minutes. Except for these symptoms his course and development were uncomplicated.

Physical examination revealed a well developed healthy boy. Blood pressure in the right arm was 155/90 mm Hg, left arm 110/90, and in the legs was unobtainable. Right brachial and both carotid pulses were normal, but no pulses were palpable in the left arm or either leg. There was a systolic murmur best heard over the base of the heart and transmitted upward into the vessels of the neck. A bruit was audible in the inter-scapular region, and there were visible and palpable collateral arteries in the superficial muscles of the back.

Röntgenograms of the chest revealed in the left upper mediastinum in the position usually represented by an aortic knob a lobulated mass which under fluoroscopy did not pulsate (Fig 22). A retrograde aortogram revealed a narrowing of the aorta distal to the left common carotid artery, and beyond this area was a lobulated aneurysmal mass filling completely with contrast medium (Fig 23).

On Aug 17, 1953, exploratory left posterolateral thoracotomy was performed, and the coarctation and aneurysm were exposed (Table IB). The sac was exceedingly thin walled on its lateral surface and the left subclavian artery originated on the posterior surface of the aneurysm about 4 cm distal to the coarctation (Fig 24). The aorta was clamped proximal to the coarctation and distal to the aneurysm, and the subclavian artery was occluded separately. The abnormal segment of aorta was excised and a segment of lyophilized homograft of aortic arch was used to bridge the defect restoring not only aortic but also subclavian arterial continuity (Fig 25). The aortic anastomoses were completed first and while blood was circulating through the aortic portion of the graft, the subclavian anastomosis was completed.

His postoperative course was uncomplicated, and he was discharged from the hospital eight days later. When last examined six months later, there were excellent pulses in all extremities. Blood pressure in the right arm was 120/60 mm Hg, left arm 100/60, left leg 130/70.

Comment—Aortic reconstruction in this case was very satisfactory in that not only was aortic continuity restored but also normal circulation to the left subclavian artery was obtained. Therefore no major vascular tributary was unnecessarily interrupted.

CASE 10—A T, a 20 year old white man, was admitted to the Methodist Hospital on Sept 13, 1953 with a diagnosis of coarctation of the aorta. At the age of 17, hypertension was discovered but he had experienced no symptoms referable to the lesion.

Physical examination revealed an extremely well developed, muscular individual in apparent excellent health. Blood pressure in both arms was 190/110 mm Hg, and in the lower extremities the blood pressure and pulses were unobtainable. A soft systolic murmur was present over the base of the heart and posteriorly a bruit was audible in the inter-scapular region. Large collateral vessels were palpable in the chest wall.



FIG. 21.—Photograph made at second operation in Case 8 showing homograft in place. Proximally the graft is sutured to lower end of another graft inserted four months previously and distally graft is sutured to abdominal aorta just above superior mesenteric artery.

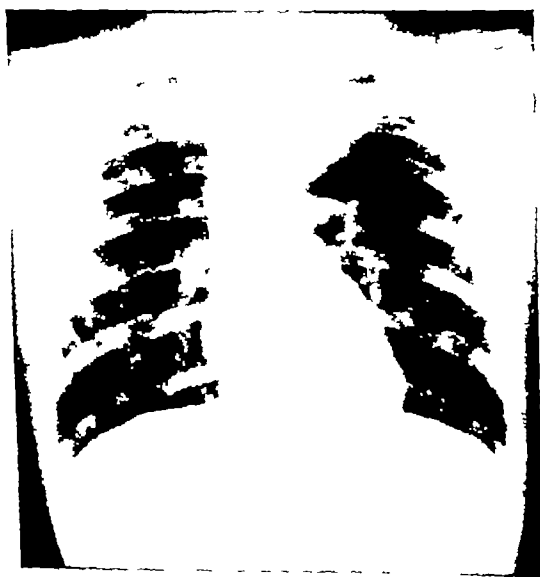


FIG. 22.—Intra rogram of chest in Case 9 showing lobulated mass in left superior mediastinum.

Roentgenograms of the chest revealed erosion of the inferior margins of many of the ribs on both sides, and the aortic knob was small.

On Sept 15, 1953, a left posterolateral thoracotomy was performed, and a long narrow coarctation distal to the level of the left subclavian artery was demonstrated measuring 3 to 4 cm in length and originating distal to the left subclavian artery (Table IB). There was an aneurysm about 3 cm in diameter near the site of coarctation and three other 0.5 cm aneurysms associated with intercostal and bronchial arteries. The segment containing the aneurysms was resected, and the 4 cm aortic defect was bridged with a lyophilized aortic homograft.



Fig. 23—Retrograde aortogram in Case 9 showing coarctation of aorta with lobulated aneurysm distal to the constriction and anomalous origin of left subclavian artery.

His post-operative progress was smooth and he was discharged from the hospital in excellent condition seven days later. When last examined on Jan 14, 1954, the blood pressure in the right arm was 140/90 mm Hg, right leg 170/100 mm Hg, and roentgenogram of the chest showed a normal configuration of the superior mediastinum.

CASE 11—J. H., a 30-year-old white male carpenter, was admitted to Methodist Hospital on Oct 25, 1953. A diagnosis of coarctation of the aorta was made in 1945 while the patient was on active duty with the Navy, but he had experienced no symptoms until the recent sudden onset of severe headache associated with vomiting and blurring of vision.

During the week prior to admission he had noticed tingling sensations in the right hand moderate stiffness of the neck and mild tinnitus. Intracranial hemorrhage was strongly suspected.

Physical examination revealed forceful pulsations in the suprasternal notch. The right radial pulse and both carotid pulses were normal but the left radial pulse as well as pulses in the legs were weak. Blood pressure in the right arm was 190/110 mm Hg left arm 140/130 and in the legs was unobtainable. The heart was not enlarged and there was a Grade II aortic systolic murmur audible to the right of the sternum in the second inter-space. Collateral vessels palpable in the chest were much more striking on the right and a pronounced systolic bruit was present to the right of the midline in the interscapular region.



FIG 24

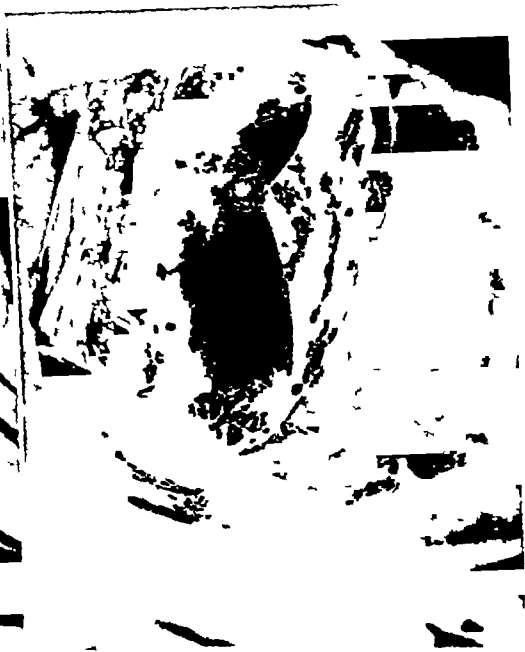


FIG 25

Fig 24—Photograph made at operation in Case 9 showing left lateral aneurysm of aorta with descending thoracic aorta extending to left. An aneurysm directed toward right in photograph is left subclavian artery.

Fig 25—Photograph made at operation in Case 9 showing aortic arch homograft in place after resection of coarctation and aneurysm. Circulation in left subclavian artery has been restored by anastomosis with corresponding vessel on graft.

Roentgenograms of the chest revealed the heart to be of normal size with absence of the normal aortic knob (Fig 26). There was deep notching of the undersurfaces of the ribs most pronounced on the right side.

Left thoracotomy was performed on Oct 27 1953 and a coarctation of the aorta 3 cm long commencing just distal to the left common carotid artery was identified (Table IB). There were two large vessels of almost equal size originating in the constricted zone one of which was probably an anomalous vertebral artery and the other the left subclavian artery. The anomalous segment of aorta was resected and a lyophilized aortic homograft was inserted in the defect. A graft of aortic arch was utilized including the left common carotid and left subclavian arteries and by this means it was possible not only to restore

aortic continuity but also to connect the two large arterial trunks to the graft (Fig 27) Upon completion of the aortic anastomoses, aortic circulation was permitted through the graft while the anastomoses of the lesser vessels were done

His postoperative recovery was uneventful, and he was discharged from the hospital nine days later He was examined five months after operation, at which time he was asymptomatic and was again working as a carpenter Pulses in all extremities were strong and the blood pressure in the right arm was 150/90 mm Hg, left arm 120/90, and left leg 130/100

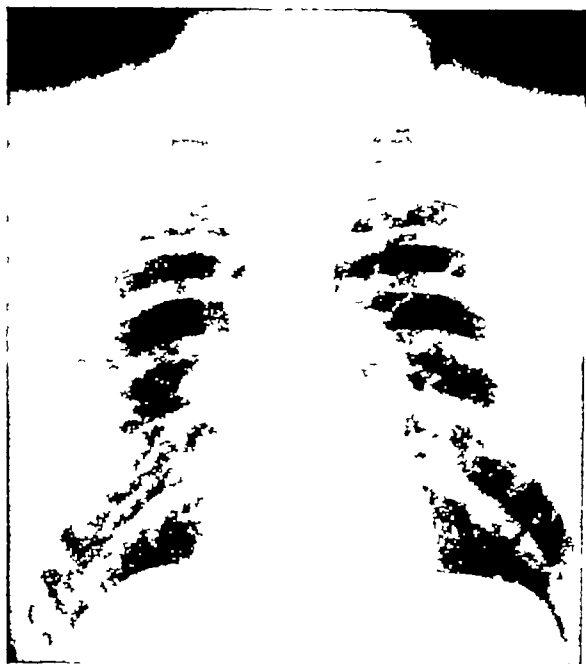


FIG. 26.—Roentgenogram of chest made in Case 11 showing absence of aortic knob in superior mediastinum and erosion of undersurfaces of ribs most striking in right hemithorax

Comment—This case is somewhat similar to another which was previously reported by Blalock⁷ in which the subclavian artery was divided and end-to-end anastomosis was possible In our case the length of the coarctation precluded end to end anastomosis The method of repair used in our case provided an ideal functional result

CASE 12—T O G, a 29 year old white woman, was admitted to Methodist Hospital on March 25 1954, with a history of hypertension for the past five years She had been asymptomatic except for occasional pounding sensations in the blood vessels of the neck Physical examination revealed blood pressure of 160/90 in the right arm, 160/110 in the left arm, and 0/0 in the right leg The pulses in the arms were equal bilaterally, but no pulses were palpable in the lower extremities The aortic second sound was markedly accentuated, and a systolic bruit was heard in the scapular region posteriorly Collateral arterial pulsations were palpable in the chest wall

Roentgenograms of the chest revealed mild cardiac enlargement with absence of the cardiac knob and notching of the undersurface of the ribs bilaterally

On March 26, 1954 at thoracotomy the coarctation was demonstrated to begin just distal to the left subclavian artery and extend for a distance of approximately 3.5 cm

with relatively little poststenotic dilatation of the aorta (Table IB) The coarctation was excised, but because of the extent of the defect a 4 cm lyophilized homograft was used to repair the defect

Postoperative convalescence was uneventful, and she was discharged from the hospital eight days later One month later the blood pressure in the right arm was 150/90 mm. Hg, left arm 146/90, right leg 150/110



Fig. 27.—Photograph made at operation in Case 11 showing on left the elongated segment of coarctation and two large arteries presumably the left vertebral and subclavian arteries arising in constricted zone. Photograph in inset shows aortic arch homograft in place and left vertebral and subclavian arteries anastomosed to stumps of left common carotid and subclavian arteries on graft respectively

SUMMARY

In fusiform aneurysms and constrictive lesions of the descending thoracic aorta, resection of an extensive segment of aorta may be necessary making end to end anastomosis impossible In such cases restoration of aortic circulation may be achieved by insertion of vascular homografts This method of therapy was employed in twelve thoracic aortic resections, seven of which were done for aneurysm and five for constrictive lesions

The degree of ischemic damage to the spinal cord occurring during periods of temporary aortic occlusion is influenced by a number of factors, including the duration of occlusion extent and location of the lesion, and extent to which collateral circulation exists In constrictive lesions the collateral circulation is usually well developed and temporary aortic occlusion may be tolerated for indefinite periods In aneurysms however, where collateral circulation is poorly developed successful performance of aortic resection depends upon prevention of ischemic damage to the spinal cord during temporary

aortic cross clamping Two cases of aneurysm are reported in which general body hypothermia was employed, and another in which local spinal cooling was used to prevent postoperative paraplegia Other means of reducing this hazard are concerned with the technical procedure itself

In constrictive lesions, where there is no haste in restoration of aortic flow, emphasis is placed upon precise reconstruction of all major vascular channels by homograft One case of acquired coarctation of the aorta is reported in which constrictions occurred in the lower thoracic aorta and in the proximal abdominal aorta Good results were obtained after two homografts were inserted to replace the diseased segment of aorta

Addendum—Since the preparation of this manuscript, 9 additional cases have been performed, 5 of which were done for aneurysms and 4 for constrictive lesions All of these patients recovered, making a total of 21 cases with 3 deaths (14.3 per cent) Of particular interest is the fact that two of the additional patients had aneurysms involving the distal part of the aortic arch similar to that described in Case 6 In both of these patients the resection and graft replacement was done under general hypothermia as described in Case 6 The proximal occluding clamp was applied just distal to the left common carotid artery in each for periods of 58 and 50 minutes, respectively Both have completely recovered with no evidence of ischemic damage to the spinal cord or other viscera

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DISCUSSION

(PAPERS BY STRANAHAN, ALLEY, SEWELL, AND KANSEL, AND COOLEY AND DEBAKEY)

DR CONRAD R LAM, Detroit, Mich—I wish to compliment the essayists of the last two papers for their very excellent application of these rather new surgical principles. It certainly requires a great deal of patience and courage. This operative work also requires a supply of homografts, and I should like to mention a method we have used for graft sterilization.

We have used a chemical named betapropiolactone* for sterilization of plasma and blood. Observation of such a sterilized graft implanted in an animal for two months shows that the graft has a perfectly normal appearance. We have observed other grafts over a period of two years. The hemotoxin and eosin stain shows the graft almost indistinguishable from the host. With trichrome stain, we see the graft side with the intimal thickening and, on the host side, we see the more normal media without intimal thickening.

In a greatly enlarged view of the normal for comparison, one notes that the muscular tissue has disappeared, leaving only the elastic tissue. This is quite typical of a perfectly functioning homologous graft.

My general surgical colleague Dr Szilagyi, has used sixteen of these sterilized grafts in human cases. He has replaced the bifurcation of the aorta four times, the common iliac twice and the femoral artery ten times. All of these grafts have appeared to serve their purpose well. The longest follow up period is six months.

DR CHARLES P BAILEY, Philadelphia, Pa—I would like to present a case which may open up another field for surgery of aneurysms. Recently we operated on a patient with a large ventricular aneurysm which extended apparently from the apex of the left ventricle to the bifurcation of the left coronary artery. The anterior descending branch was obliterated by the occlusion, producing the infarction which was the cause of this aneurysm. As you know, ventricular aneurysms nearly always are caused by infarction and are said to occur in about 15 per cent of all acute coronary occlusions. Because pathologic study of these lesions indicates that these aneurysms have very tough fibrotic bases, and since they rupture at the fundus rather than at the base, we thought it would be safe to put a clamp across the base of the aneurysm, cut it off, and oversew it. The largest clamp we had the double length Satinsky clamp, only extended across about two thirds of the aneurysm so we placed large sutures in continuous mattress fashion, through the base of the sac from one extremity of the ventricle to the other. Then by traction on the extremities of the sutures the loops became straightened out and pulled the entire base together in a straight line. Then we placed another row of interrupted mattress sutures. Finally the sac was amputated and the cut edges were oversewn with a third row of sutures which we thought would be good for reinforcement. There were clots in this region although near the apex of the ventricle there were none.

Actually this is not the first such operation. Dr Robert Glover performed a similar operation almost one year ago, and I am sure it would have been successful except that the patient bled to death from a gastric ulcer several days after surgery. It appeared good at autopsy. There may have been other similar cases of which I am not aware. Of course we know about the work of Drs Murray, Allen, and Beal in the experimental resection of infarcts, which was fundamental to this concept. They were attempting to operate on acute infarctions of the heart, however, which I think would be much more risky.

This patient, who preoperatively had heart failure, shortness of breath, and gallop rhythm that could not be relieved medically, on the operating table developed a higher blood pressure and better cardiac action as soon as this paradoxically dilating aneurysm was removed. He has had no gallop rhythm and no dyspnea since the operation. I think that this technique might be applied rather generally in those patients who develop aneu-

*Obtained from the B F Goodrich Research Laboratories Brecksville Ohio

risms following myocardial infarction if they show evidence radiographically of progressive enlargement and impending rupture or present severe disability. The pathology would seem to indicate that the sutures will hold well and this clinical experience suggests that they may become greatly improved.

DR HENRY T. BAHNSON, Baltimore, Maryland—I enjoyed both these presentations and have only one point I would like to make that is that a graft is not always required. Syphilis and trauma usually cause localized aneurysms and frequently one may perform an aortorrhaphy without the use of a graft. We have required a shunt and a graft in only one of the sixteen thoracic aneurysms that we have treated surgically; this one was treated unsuccessfully, however. Certainly the contributions which have been made in the form of shunts and hypothermia will add to the safety of inserting grafts. I think the principle of a lateral aortorrhaphy is valid particularly with traumatic aneurysms, because in order for a patient to survive a traumatic aneurysm the tear almost has to be a fairly limited one. The two shown here occurred in the typical position for a traumatic aneurysm. The common cause at present is an automobile accident and not always with the split pea and walnut syndrome.

DR JAMES FINNERTY, New York, N. Y.—I would like to make a comment about the importance of maintaining collateral circulation in coarctation of the aorta. Two years ago we had the unfortunate experience of resecting the coarctation in a patient who had an innominate artery that originated from the distal aorta immediately beyond the coarctation and, not having recognized this anomaly preoperatively, in order to gain strength we divided the innominate artery. This patient postoperatively had a transient left hemiplegia, absent pulse in the right arm and absent right carotid pulse. The left hemiplegia cleared, but the patient was left with persistent paraplegia. I think if we bear this in mind we might occasionally avoid this complication.

One aneurysm of the ascending aorta was present in a rather unusual position, namely on the left side. The angiogram failed to show increased opacification of the aneurysm. At operation it was possible to do a lateral aneurysmorrhaphy, as suggested by Dr. Bahnson, and the patient has done well since.

LT WILLIAM H. SEWELL (Closing)—The decision to use a heterograft in the first case was based largely on some work done at the Naval Medical Research Institute at Bethesda.

Studies were carried out in which aqueous extracts were made of both fresh and freeze-dried arteries. Rabbits were immunized with these extracts and later were challenged with the same antigen. All the rabbits that were immunized with the fresh extract and challenged with the same one showed a positive precipitin test; those that were immunized with the extract of the freeze-dried artery and challenged with that showed no precipitins. The ones in which one extract was used as immunizing antigen and the other for the challenge and vice versa showed a positive precipitin in some of the animals.

We concluded that these findings may well explain the fact that there was less foreign body reaction in the freeze-dried grafts than there was in the fresh in our series. There was less fibrosis around the freeze-dried grafts. They lost elasticity more rapidly but soon leveled off, whereas the fresh grafts continued to lose it for a long period of time, and ended up much less elastic.

In the freeze-dried grafts the muscle degeneration occurs much more rapidly than in the fresh and replacement by fibroblasts also occurs more rapidly and is more nearly complete.

The difference between the fresh and freeze-dried homografts was not very great. The freeze-dried heterografts, however, transplanted from the pig to the abdominal aorta of the dog, showed that the results were almost as good grossly as those of the fresh homograft. This is certainly a much better result than would be obtained if we simply took a fresh heterograft from a pig and put it into the dog. We feel that heterografts are not preferable to homografts, and the reason it was used was because the man's aorta above

and below the aneurysm was so dilated that it was impossible to fit it with a homograft. The graft was taken from two calves, and even so it was smaller than the aorta, but it was a much closer fit than could have been obtained with a human vessel. The calf aorta is two to three times the diameter of the human aorta.

I think that plastic cloth would be satisfactory for most types of aortic grafting, but in an area where there are large branches coming off, I suspect that the turbulence which would occur would give propagation of the thrombus lining the inside of the cloth and probably would cause trouble.

DR M E DEBAKEY (Closing)—I should like to express our appreciation to the men who have discussed this paper. With reference to a point made by Dr Bahnson regarding lateral aortorrhaphy, we too are convinced that it is often the procedure of choice. That is particularly true in syphilitic aneurysms where there is a relatively small sacciform type of aneurysm. This procedure, as Dr Bahnson and those interested in it know quite well, was first developed by Dr Tuffier more than fifty years ago, and the principles of the operation were clearly described by him although he himself was never successful in applying it.

So far as heterologous grafts are concerned, our experimental observations lead us to feel that they are not as satisfactory as homografts, to the extent that we would be very hesitant to use a heterograft in a human patient. So far as the procedure of shunts is concerned, we realize that this may have definite application in the resection of the arch where the carotids and innominate artery may have to be occluded for a significant period of time. The success we had with the first patient, however, on whom we reported about a year ago, in resecting an aneurysm of the descending thoracic aorta without the use of a shunt, made us feel that to introduce this procedure would complicate the operation if it was not absolutely essential. For this reason we are inclined to feel that in the resection of aneurysms involving the descending thoracic aorta up to the level of the subclavian, with the use of hypothermia of moderate degree, and the expedition of the operation so that the occlusion time is not beyond an hour, it is possible to do this operation quite successfully without the use of shunts and without complications.

Finally, one point I would like to make is with regard to the ultimate fate of homografts. Dr Gross, as you know, has had an extensive experience with the use of grafts for a longer period, perhaps, than anyone else, and has found that they have stood up well, certainly for five or six years. We have had the fortunate opportunity to excise pieces of graft after varying periods up to one year, and the study of these grafts has shown that they behave very much as experimental observations would indicate that they would behave, and at the end of one year their architectural structure is still functionally satisfactory. For this reason we are inclined to believe they will provide satisfactory results for a much longer time.

THE ARTERIAL OXYGEN TENSION AFTER RESECTION WITH SIMULTANEOUS OSTEOPLASTIC THORACOPLASTY

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THE aim of treatment for pulmonary tuberculosis is to achieve persistent negative guinea pig tests of the sputum and gastric washings with a maximum preservation of pulmonary function. To achieve this result, a combination of antimicrobial therapy, bed rest, resection, and thoracoplasty must be used. The treatment must be individualized for the cases with pulmonary tuberculosis. In a large group of cases however, it is obvious that all four therapeutic measures must be utilized. In this group we consider a resection with simultaneous osteoplastic thoracoplasty the treatment of choice.

A preliminary thoracoplasty with secondary resection has several disadvantages

- 1 When the patient recovers from the first operation he has to look forward to a second major one
- 2 The danger of spread is greater after a thoracoplasty than after a resection
- 3 The hospitalization of the patient is prolonged from three to six months
- 4 It is more difficult to model the chest in the desired size at a second operation

The only advantage with the two-stage procedure was that there was no paradoxical movement at the second operation. The resection was therefore considered safer when the thoracoplasty had already been performed.

The new osteoplastic thoracoplasty according to Bjork^{1,2} can be made in any desired size and will make a stable roof for the thorax when the resection is completed. As the paradoxical movements are excluded the patients will stand this combined procedure very well. In the first one hundred cases of resections with simultaneous osteoplastic thoracoplasty, only one was lost.² The operation is only prolonged by thirty to forty-five minutes. The patients need however three or four extra blood transfusions, as usually 1,500 to 2,000 ml of bloody fluid is drained off during the first forty-eight postoperative hours. In this paper we intend to show the arterial oxygen tension in the post-operative course after resections with simultaneous osteoplastic thoracoplasty as compared to resection under a preliminary thoracoplasty.

MATERIAL

Thirty consecutive patients with pulmonary tuberculosis undergoing resection with simultaneous osteoplastic thoracoplasty were investigated (Group

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This work was aided by The Swedish National Association against Tuberculosis.
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I) For comparison, twenty-nine patients with pulmonary tuberculosis with a preliminary thoracoplasty were investigated when undergoing resection at a second stage (Group II)

METHOD

With an indwelling needle in the femoral artery, a blood sample was taken at rest after the patient had been hyperventilating as much as possible for about twenty-five seconds. Oxygen and carbon dioxide tensions (pO_2 and pCO_2) in the arterial blood were determined according to Riley, Proemmel, and Franke⁷ with a slight modification. The saline or saline-Dreft solution has been omitted for washing the capillary. The sodium hydroxide solution is applied for both the capillary washing and the simultaneous carbon dioxide combining, thereby eliminating one source of error.³ This investigation was performed before operation and at intervals during the postoperative period.

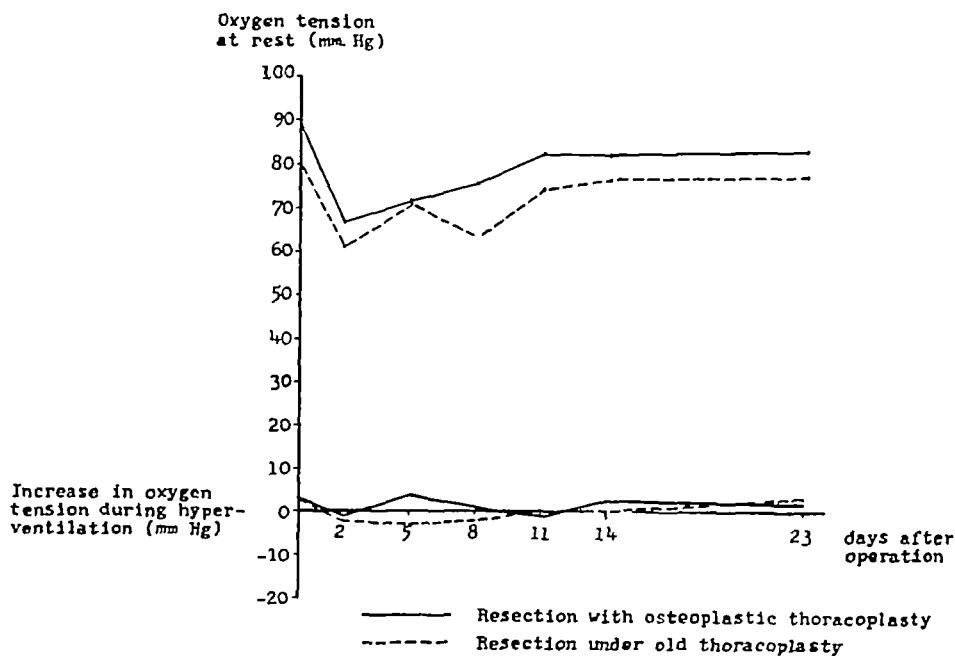


Fig. 1—The two curves for resting arterial oxygen tension are on the whole parallel whether the resection is done with simultaneous osteoplastic thoracoplasty or under an old thoracoplasty.

RESULTS

The investigation has shown a decrease of the resting arterial oxygen tension during the postoperative course of the same magnitude in the two groups (Table I Table II and Fig. 1). Thus the two curves for resting arterial oxygen tension are on the whole parallel, whether the resection is done with simultaneous osteoplastic thoracoplasty or under an old thoracoplasty. Nor

has it been possible to see any significant difference in the two groups in the arterial oxygen tension during voluntary hyperventilation (Table I, Table II, and Fig 1)

DISCUSSION

The stable roof of the thorax obtained by the osteoplastic thoracoplasty will exclude paradoxical respiration and facilitate an effective cough. Thus no more cases of atelectasis are found if this thoracoplasty is added at the same time as a resection as compared to the group of resections under primary thoracoplasties.

The investigation of the resting arterial oxygen tension during the postoperative period has confirmed the clinical impression that the osteoplastic thoracoplasty is a physiologic operation that can be added to a resection when indicated without hesitation from a functional point of view.

TABLE I RESECTION WITH OSTEOPLASTIC THORACOPLASTY

	BEFORE OPERATION	DAYS AFTER OPERATION					
		1 3	4 6	7 9	10 12	13 15	16 30
<i>Resting Oxygen Tension (mm Hg)</i>							
Number of cases	30	15	14	15	11	19	8
Mean value	89.1 ± 1.0	66.8 ± 2.3	71.9 ± 2.1	75.9 ± 1.9	82.4 ± 1.3	82.1 ± 1.4	82.7 ± 2.0
Standard deviation	5.6	8.9	7.8	7.5	4.3	6.0	5.8
<i>Increase in Oxygen Tension (mm Hg) During Hyperventilation</i>							
Number of cases	29	14	13	15	10	19	8
Mean value	3.0 ± 1.3	-1.0 ± 1.7	+3.7 ± 1.9	+1.1 ± 1.6	-0.3 ± 1.7	+2.8 ± 1.2	+2.0 ± 3.1
Standard deviation	6.7	6.5	6.7	6.2	5.4	5.3	8.8

TABLE II RESECTION UNDER OLD THORACOPLASTY

	BEFORE OPERATION	DAYS AFTER OPERATION					
		1 3	4 6	7 9	10 12	13 15	16 30
<i>Resting Oxygen Tension (mm Hg)</i>							
Number of cases	31	6	5	7	4	17	6
Mean value	80.2±1.0	61.4±5.2	70.7±3.1	63.5±3.0	74.2±1.0	76.3±2.2	76.0±3.9
Standard deviation	10.5	12.8	6.9	8.0	1.9	8.9	9.5
<i>Increase in Oxygen Tension (mm Hg) During Hyperventilation</i>							
Number of cases	31	6	5	7	4	17	6
Mean value	2.8±1.5	-1.4±1.7	-3.3±3.2	-2.2±2.4	+0.2±2.3	+0.3±2.3	-2.1±1.3
Standard deviation	8.2	4.3	7.2	6.3	4.6	9.6	3.1

A patient's ability to increase the arterial oxygen tension during voluntary hyperventilation in the immediate postoperative period will partially depend on the pain. The increase of oxygen tension during hyperventilation in patients undergoing resection with osteoplastic thoracoplasty has not been less than in cases with resection under old thoracoplasties. We believe this confirms the clinical impression that the patients with the combined operation have no more pain than patients with the two stage procedure.

SUMMARY

The arterial oxygen tension at rest and during voluntary hyperventilation has been investigated during the early postoperative course in thirty pa-

tients undergoing resection with simultaneous osteoplastic thoracoplasty. The results have shown this to be a well-tolerated procedure and that there is no reason from a functional point of view to perform resection with thoracoplasty in two stages.

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Original Communications

THE CLINICOPATHOLOGIC SIGNIFICANCE OF THE DEMONSTRATION OF VIABLE TUBERCLE BACILLI IN RESECTED LESIONS

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ONE year ago, O'Brien and associates¹ presented before this Association a report on the chaos regarding the resection of residual caseous lesions following chemotherapy. These authors concluded that too many necrotic lesions were resected. They also stated that some investigators were raising the question as to whether closed lesions should be resected.

The great wave of resectional surgery in tuberculosis may be attributed to several factors. (1) As a result of prolonged chemotherapy in cases of pulmonary tuberculosis soon after its origin, the disease undergoes extensive resolution, and the process is limited to a portion of a lobe or lung. (2) As a result of technical advances in thoracic surgery, portions of the lung can be removed with much greater facility.

D'Esopo and co-workers² at the Veterans Administration Hospital, Summit, N Y, first reported that after the prolonged use of streptomycin and PAS, cavities closed and sputum conversion occurred in a high percentage of cases.

Medlar, working with this group at that time, urged the surgeons to remove as many of these lesions as possible. He felt that it was important to learn what effect chemotherapy was having on the lesions and also raised the question as to whether it was safe to leave many of these foci in the lung. He reasoned that many of these foci were in communication with bronchi and would therefore be a potential source of danger if left behind.

Ryan, Medlar, and Welles³ subsequently reported on the excision of necrotic foci in the lungs following the prolonged use of streptomycin on a regimen of 1 Gm daily and PAS 12 Gm daily.

From the Veterans Administration Hospital, East Orange, N J and the Research Division of Chas. Pfizer & Co. Inc., Brooklyn, N Y.
Read at the Thirty-fourth Annual Meeting of the American Association for Thoracic Surgery, Montreal, Quebec, May 3, 4, and 5, 1954.

Subsequent to these reports, necrotic foci of 1 mm or more were removed from the lungs of many patients who received chemotherapy for varying periods of time

D'Esopo and co workers⁴ have demonstrated that the tubercle bacilli present in the necrotic foci after long-term chemotherapy in most instances cannot be cultivated *in vitro* by standard microbiologic techniques Medlar and associates⁵ obtained positive cultures in only 7 per cent of the closed lesions from the same institution Steenken and Wolinsky,⁶ who utilized more varied and specialized procedures for the purpose, confirmed this observation Steele⁷ has published analogous observations Similar bacteriologic findings have been reported by Granville and associates⁸ In a group of 12 patients who received streptomycin and PAS (all but 2 received streptomycin 1 Gm daily for a period of from 21 days to 495 days) and in whom the cavities closed, they found viable tubercle bacilli in only 2 of the 12 resected tissues The presence, however, of milary and conglomerate tubercles outside the capsule of the nodular caseous lesions suggested to these authors that there were viable organisms in these lesions The conclusion has been reached that these organisms "behave as if they were dead"⁹

Thus two extremely important questions arise in relation to excisional surgery (1) Are all necrotic foci potentially dangerous? (2) Are viable tubercle bacilli present within the necrotic foci?

The necrotic foci present after long-term chemotherapy are of two types which, although they may be distinguished microscopically, are not always separable on gross examination It is our opinion that, although these foci resemble each other closely they hold different potential dangers The first is the necrotic focus which has never undergone liquefaction, the second represents a tuberculous cavity in which the lumen has been obliterated by an inspissation of its necrotic fragments

In both types the process begins as a tuberculous pneumonia which soon undergoes necrosis Generally, the smaller the original area of tuberculous pneumonia, the greater is the opportunity for healing and encapsulation to develop Cavity formation in necrotic foci less than 1 cm in diameter is extremely infrequent, and occurrence in foci less than 5 mm is rare indeed Not all of the larger necrotic foci on the other hand, undergo liquefaction and cavitation

In the development of encapsulated caseous foci, the zone of tuberculous pneumonia undergoes necrosis, and a zone of granulation tissue encloses it completely The elastic framework in the necrotic tissue remains intact since the original process was that of a tuberculous pneumonia in which there was no dissolution as a result of liquefaction As healing progresses, the encircling zone of granulation tissue is gradually transformed into hyalinized connective tissue, the contracting capsule results in a decrease in the size of the central necrotic area It has been our experience that these foci do not communicate with bronchi because the capsule around the necrosis is complete and delimits it from the surrounding lung tissue We have had the oppor-

tunity in this series to serial-section many such necrotic foci of various size. In no single instance have we observed a bronchus communicating with the necrotic mass.

We believe that the necrotic foci which have never liquefied and have subsequently become encapsulated are not a potential source of danger. The reason for this is twofold: (1) they do not communicate with bronchi, and (2) they do not subsequently undergo liquefaction, but become progressively firmer with the deposition of calcium salts.

The process is entirely different in inspissated cavities. The progression to cavitation from a tuberculous pneumonia generally occurs through the invasion of the zone of necrosis by polymorphonuclear leukocytes. Following softening, liquefaction, and evacuation of much of the liquefied material, a cavity results within which necrotic fragments of various size are present both in the lumen and attached to the inner wall. The cavity empties its contents into a draining bronchus, the latter is ulcerated as it enters the cavity (when no chemotherapy has been used). In cavity closure, as the lumen of the cavity decreases, the individual necrotic fragments, which had been separated by the process of liquefaction, are compressed into the narrowed space. Thus even after calcium salts are deposited, the necrotic material remains as fragments in the enclosed space. Microscopically, although a few intact elastic fibers are still observed in some of the necrotic fragments, the majority of them show a great loss of elastic fibers. Such cavity closure, occurring after chemotherapy, shows re-epithelization at the bronchocavitary junction. The epithelium from the bronchus extends along the inner wall of the cavity for a variable distance.

Comparing the healed cavity after chemotherapy with that healed without chemotherapy, it is found that in treated cases the bronchus is patent and re-epithelized, while in untreated cases no bronchial communication can be demonstrated. As a result of the communicating bronchus the inspissated contents may subsequently be evacuated. Thus, open healing is a much more frequent occurrence following chemotherapy. A consequence of the evacuation of much of the necrotic contents within the inspissated cavity will be an extremely small cavity.

In a previous study of the bronchocavitary junction following chemotherapy we raised the question, "Is the inspissated cavity with a patent bronchus a potential source of danger?"¹⁰ One of the most important reasons for raising the question at that time was that we observed secondary liquefaction of the inspissated necrotic contents within the lumen of the cavity. Neutrophils intermingled with mucin secreted into the bronchial lumen were observed to extend among the calcified necrotic fragments and result in an evacuation of this material. We stated then that it would depend upon the fate of tubercle bacilli in the inspissated cavity in patients undergoing prolonged combined chemotherapy. D'Esopo and associates¹¹ have described the effects of long-term combined therapy on bacilli in such lesions. They found that, in patients treated for eight months or longer with daily streptomycin-PAS in whom the cavity disappeared from roentgenographic view, they could neither grow cultures of tubercle bacilli nor produce disease in the guinea pig inoculated with

contents of excised inspissated cavities in a high percentage of cases (1 of 36 cases). Smears from these lesions invariably showed acid-fast organisms, often in great numbers. These writers concluded that in such lesions the tubercle bacilli are probably not viable. Falk and associates¹² believed that their findings confirmed the views expressed by D'Esopo and his co-workers.

We have previously stated that if the bacilli in the inspissated cavities of streptomycin-treated patients should prove to be viable after discontinuation of therapy, it would seem hazardous to leave such lesions in situ.¹⁰ The possibility must be considered that, if the bacilli were again to multiply within these inspissated lesions, reactivation of disease would occur. Such patients subjected to re-treatment would be expected to develop streptomycin resistance rapidly. Should bacilli remain viable within residual inspissated lesions, it would be imperative that the indications for prolonged chemotherapy be carefully evaluated.

We concluded then that the potential dangers inherent in such inspissated lesions with patent, re-epithelized draining bronchi have not yet been precisely defined. As a consequence, it is not yet possible to determine whether such obliterated residual cavity foci should be left in situ or whether resection of such areas is obligatory.

The possibility that more refined cultural techniques might permit the demonstration of viable tubercle bacilli in necrotic tuberculous lesions has been discussed by various investigators.¹³⁻¹⁷ In a recent communication,² Hobby and associates²⁸ have summarized a portion of the evidence which suggests that microorganisms at times are capable of survival for long periods of time under adverse environmental conditions. These investigators have shown also that, by use of special cultural conditions and long-term incubation, growth of tubercle bacilli can indeed be demonstrated in cultures of resected necrotic lesions.

The present study was undertaken in an attempt to correlate the anatomic findings with the viability of tubercle bacilli in resected lesions from patients who had had extensive chemotherapy, and who had been culture negative for several months. The microbiologic observations and the methods used are presented in detail in another report²⁸ while the clinicopathologic nature of the material is reported herein.

Material—As of this time necrotic fragments from 40 patients have been cultured. Included in this study are inspissated cavities, necrotic foci which have never liquefied, and also necrotic fragments from cavities showing open healing.

In selecting the cases for study an attempt was made to meet as closely as possible the criteria set up by D'Esopo and his associates. The high percentage of negative results obtained by these authors were in cases where there was a closure of all cavities, consistently negative sputum and gastric cultures for several months and stability of x-rays. They emphasized that the results obtained were in a highly selected group and also that this was a study. They viewed their material as a gathering of bacteriologic data and not for their clinical application in so far as surgical procedures are concerned.

Because of the long observation period needed for evaluation of results, only the first 19 cases are included in this report. Eleven of these 19 studies were original treatment cases, four were retreated cases and four were controls.

CASE REPORTS—ORIGINAL TREATMENT CASES

CASE 1—J. R. was a 29-year-old Puerto Rican man with bacteriologically proved pulmonary tuberculosis and a small cavity in the superior segment of the left lower lobe by x-ray. Chemotherapy was initiated on March 4, 1953, in a dosage of 1 Gm. streptomycin daily with 12 Gm. PAS daily. This regimen was continued without interruption with the exception that, on March 19, 1953, the regimen was changed to streptomycin 1 Gm. twice weekly with PAS, 12 Gm. daily. All smears and cultures of sputum concentrates and gastric specimens were negative from April 27, 1953 on. X-rays showed marked regression of the lesion in the left lower lobe with no evidence of cavity on left lateral tomogram preoperatively and with stability for at least 3 months preoperatively. On July 13, 1953, a segmental resection of the superior segment of the left lower lobe was performed.

Pathologic Findings—The specimen of the superior segment of the left lower lobe measures 7.5 by 6 by 3 cm. Occasional yellow necrotic foci varying from 2 to 3 mm are present in an otherwise resilient lung parenchyma.

Microscopically an occasional necrotic focus is present. One of these proved to be an inspissated cavity while the others are encapsulated caseous foci which had never liquefied. Serial sections show a bronchus opening into the inspissated cavity. The walls of the inspissated cavity as well as the capsules around the necrotic foci which had never liquefied are composed of hyalinized connective tissue with occasional epithelioid and giant cells. Occasional epithelioid giant cell foci without a necrotic center are also present.

Bacteriologic Findings—A single 2 mm. necrotic focus was removed in its entirety for culture. This specimen was negative for acid fast bacilli both on smear and culture.

CASE 2—E. H. was a 47-year-old white man with proved pulmonary tuberculosis involving the right upper lobe. Streptomycin 1 Gm. twice weekly, and PAS 12 Gm. daily were initiated on Oct. 1, 1952 (Fig. 1 A) and were continued without interruption. All specimens taken after start of chemotherapy were negative by culture. Four sputum concentrates between Dec. 31, 1952 and Feb. 9, 1953 (Fig. 1 B) were smear positive for acid fast bacilli while a single specimen taken Feb. 24, 1953, was negative by smear as well as by culture. The patient revealed the greatest resolution of disease roentgenographically between August and December 1952. At the time of surgery tomograms revealed extensive involvement of the entire right upper lobe region with multiple small areas of radiolucency. X-rays indicated roentgen stability since December 1952. The right upper lobe was resected on March 19, 1953 (Fig. 1 C). X-ray of the inflated resected right upper lobe showed only a few small radiolucencies within a large dense lesion (Fig. 1 D).

Pathologic Findings—The specimen of the right upper lobe measures 14 by 12 by 4 cm. Present in the midaspect of the lobe are many oval and round yellow, necrotic foci arranged singly and in contiguous groups. These foci vary in size from 2 mm. to 3 cm. (Fig. 1 E). The smaller foci have a smooth yellow appearance. The larger ones, representing inspissated cavities, have a fragmented appearance. Branches of the bronchi open widely into these cavities.

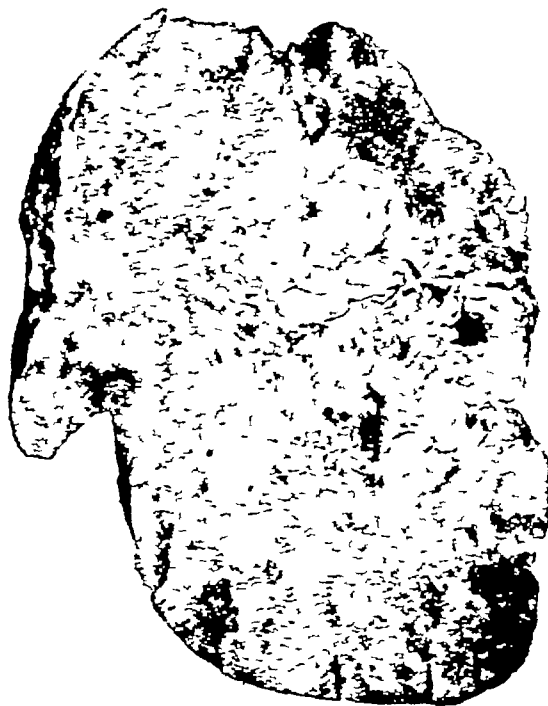
Microscopically, the inspissated cavities are filled with fragmented, necrotic fragments containing calcium salts. The cavity wall is composed of granulation tissue in some aspects but in most regions is made up of dense hyalinized connective tissue. Branches of the bronchi open into the cavity (Fig. 1, F). There is re-epithelization of the bronchus at the bronchocavitary junction. The lumen of the bronchus is filled with necrotic material similar to that present in the inspissated cavity. The smaller necrotic foci, which show the ghost forms of the underlying lung structure, are surrounded by narrow connective tissue capsules (Fig. 1, G).

Bacteriologic Findings—Part of a large inspissated cavity was removed for bacteriologic observation, acid fast smears of this specimen showed small numbers of bacilli while cultures were positive for tubercle bacilli. A filled in cavity with necrotic foci, although negative on smear was culture positive for acid fast bacilli.

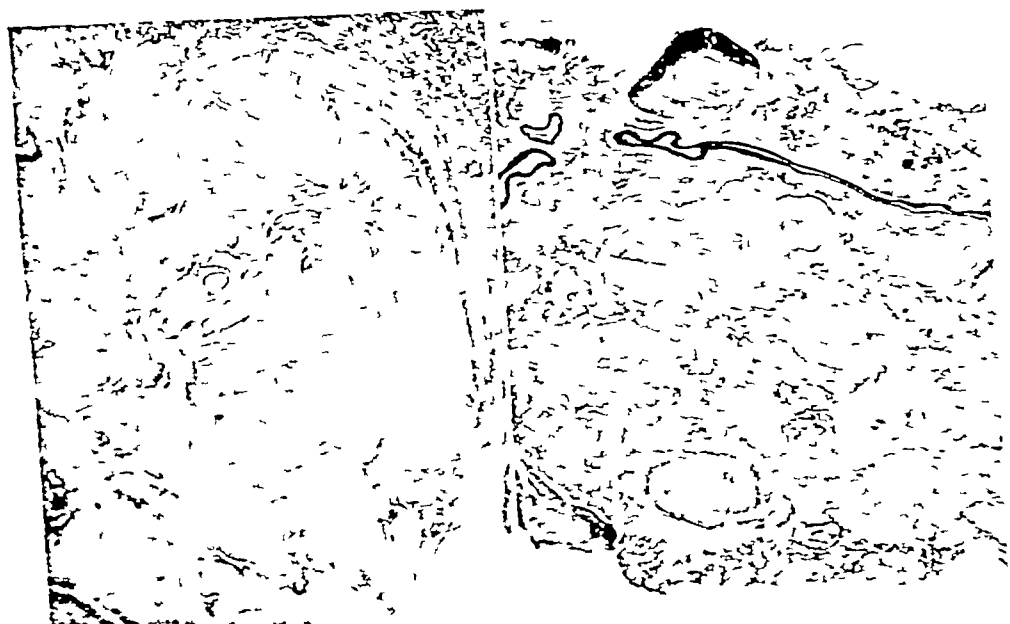


FIG. 1. A-D—CASE 2.—A Chest X ray Jan. 3, 1953. Pulmonary tuberculosis had been discovered August 1952 and streptomycin and PAS therapy begun Oct. 1, 1952. B Anteroposterior tomogram Feb. 4, 1953. At this time cultures had been negative for almost four months but sputum smears were still positive. C Right lateral tomographic view Feb. 18, 1953. Right upper lobectomy was done March 19, 1954. D X ray of inflated resected right upper lobe. On section this specimen showed necrotic foci and filled in cavity (3 mm. to 3 cm.).

CASE 3—E. C. was a 57-year-old white man with diabetes whose illness began in August 1952 with a spontaneous right pneumothorax and a pleural effusion. Following re-expansion of the right lung a lesion thought to be cavitory was seen in the right lower lobe. Streptomycin 1 Gm. twice weekly, and PAS 12 Gm. daily, were initiated on Dec. 7,



E



F

G

Fig. 1. *E*—Coronal section of lung showing a number of inspissated material and small necrotic foci. *F*—Photomicrograph of a large inspissated cavity and a bronchovascular junction is put at and re-epithelized. *G*—Photomicrograph of a small necrotic foci showing thin connective tissue capsules. The thin connective tissue is among the tuberculous foci.

1952, and continued without interruption. Pleural fluid at time of the pleural effusion was negative on culture, although sputum was smear positive for acid fast bacilli. Following start of chemotherapy, sputum concentrates were persistently negative on culture from Nov. 5, 1952, until time of surgery, although positive on smear until after March 16, 1953. Decortication of the right lung and a lobectomy of the right lower lobe was performed on April 27, 1953. Section of the resected specimen revealed a small inspissated cavity in the superior segment of the right lower lobe, not corresponding to the lucency seen on tomograms, with adjacent 2 to 5 mm necrotic foci. It was concluded that the tomographic conformation, which had been interpreted as a cavity, actually was due to the pleural changes following resorption of fluid and re expansion of the right lung.

Pathologic Findings—The specimen measures 9.5 by 8 by 8.5 cm. Present throughout the lobe, particularly in the superior aspect, are groups of oval and round, yellow, necrotic foci varying in size from 2 to 5 mm surrounded by a narrow gray capsule. Some of the larger ones show fragmentation of the necrotic contents.

Microscopically, some of the larger foci show fragmentation of the necrotic fragments in which there is a loss of lung architecture. Among the necrotic fragments are also blue zones resembling mucin. Serial sections show a patent epithelized bronchus opening into the cavities. The smaller necrotic foci and inspissated cavities are surrounded by narrow hyalinized connective tissue capsules containing small numbers of fibroblasts and epithelioid cells.

The smaller necrotic foci show a preservation of the elastic framework and are surrounded by hyalinized connective tissue containing a few fibroblasts and epithelioid cells.

Bacteriologic Findings—A specimen containing hard, necrotic foci showed moderate numbers of acid fast bacilli on smear and was positive on culture. The soft liquefied necrotic material from an inspissated cavity, although positive on smear (1 to 2 acid fast bacilli per microscopic field), was negative on culture. The wall of this cavity, in contrast, showed viable tubercle bacilli on culture despite the fact that acid fast bacilli were not detected on prolonged examination of smears.

CASE 4—I. D. R. was a 44 year old Portuguese man with a history of pulmonary tuberculosis with a right upper lobe cavity since 1946. Streptomycin, 1 Gm daily, and PAS, 12 Gm daily, were started on July 20, 1952, and continued without interruption, with the exception that after Nov. 20, 1952, streptomycin was administered in a dosage of 1 Gm twice weekly. All cultures were negative with the exception of one specimen taken on Nov. 21, 1952. Three of 12 sputum concentrates were positive on smear between Nov. 20, 1952, and March 9, 1953, the last smear positive specimen was taken on Jan. 12, 1953. No significant roentgenographic change was noted during the four months prior to surgery. Cavity was lost to view on conventional films six months prior to surgery, and on tomograms two months prior to surgery. On March 4, 1953, a right upper and right middle lobectomy was performed. X ray of the inflated resected lobes revealed no open cavity.

Pathologic Findings—The specimen consists of the right upper and right middle lobe, which together measure 12 by 11 by 6 cm. Occasional necrotic foci present are limited to the superior portion of the upper lobe. Most of the foci have a chalky appearance. One of the larger ones is fragmented, but no communication with a bronchus can be demonstrated.

Microscopically most of the necrotic foci show the elastic fibers within the necrotic zones to be intact. Cholesterol deposits and calcium salts are present within the zones of necrosis. The capsules are composed of hyalinized connective tissue containing only occasional fibroblasts and epithelioid cells.

Serial sections reveal a re epithelized bronchus opening into the inspissated cavity. Some of the sections show irregular necrotic fragments in the lumen of the bronchus. The cavity wall, which is composed of hyalinized connective tissue, in occasional sections shows a few epithelioid and Langhans' giant cells.

Bacteriologic Findings—A portion of the necrotic contents of the inspissated cavity in the right upper lobe was negative for acid fast bacilli both on smear and culture. Putty like foci at the base of the same lobe likewise were negative on culture, although a few sparsely scattered acid fast bacilli were observed on smear.

CASE 5—F. K. was a 34 year old white man in whom a nodular lesion in the left lung was discovered in June, 1952, in the presence of a left pleural effusion. Streptomycin, 1 Gm twice weekly, and PAS, 12 Gm daily, were started on Sept. 18, 1952, and continued without interruption. Thoracentesis in June, 1952, with inoculation of obtained fluid into guinea pigs, resulted in positive identification of tubercle bacilli. All sputum and gastric specimens were negative on smear and culture from June, 1952, to April, 1953, with the exception of one isolated positive sputum culture on April 9. This single positive sputum culture was followed by negative smears and culture on a single tracheal lavage specimen taken May 5, 1953. X-ray on Jan. 12, 1953, revealed a solid lesion 2 cm in diameter in the lateral aspect of the left mid lung field and obliteration of the left costophrenic angle. There was no change in the size or character of the left lung infiltrate between June, 1952, and the time of resection as evidenced by serial roentgenograms, and it was thought that the infiltrate represented an encapsulated caseous focus. On May 12, 1953, a wedge resection from the anterior segment of the left upper lobe was performed. X-ray of the resected specimen showed only a small dense nodule which on sectioning proved to be a 2 cm cavity with liquefied contents.

Pathologic Findings—The wedge of tissue measures 3 by 3.2 by 2 cm. The lung tissue contains several necrotic foci varying in size from 2 to 5 mm. The largest focus contains liquefied yellow material. Narrow gray capsules enclose the necrotic zones.

Microscopically the necrotic foci which show the ghost forms of the underlying lung structure are surrounded by wide areas of hyalinized connective tissue containing epithelioid and giant cells.

Serial sections of the cavity containing the liquefied contents show a patent bronchus opening into the inspissated cavity. Numerous neutrophils are intermingled among the necrotic fragments of the cavity lumen.

Bacteriologic Findings—Half of the inspissated cavity including a portion of the wall and of the liquefied contents, was removed for bacteriologic study. Cultures were negative even after prolonged incubation, despite the presence of innumerable acid fast bacilli on smear.

CASE 6—T. H. was a 35 year old white man with bacteriologically proved pulmonary tuberculosis since April, 1952. Isoniazid 300 mg daily was administered for 120 days beginning April 22 and terminating Aug. 19, 1952. Streptomycin 1 Gm twice weekly, and PAS, 12 Gm daily, were initiated on Aug. 25, 1952, with pneumoperitoneum, and continued without interruption until the time of surgery. All sputum cultures beginning with December, 1952, were negative on smear and culture with the exception of one specimen taken Jan. 5, 1953, which was positive on culture. Four specimens taken subsequent to January 5 and prior to surgery were negative both by smear and culture. Serial films between May 9 and Aug. 17, 1952, showed no essential change although there appeared to be some resolution and hardening of lesions between April 14 and June 9, 1952. Subsequent films between Aug. 15 and Nov. 14, 1952, revealed considerable retraction in the right upper lobe as well as some clearing of small densities in the left apex. Anteroposterior tomograms on Jan. 29, 1953, revealed linear and nodular densities with "honeycombing" present in the shrunken right upper lobe. Bilateral lesions had been stable since November 1952. A resection of the apical and posterior segments of the right upper lobe was performed on Feb. 26, 1953. X-ray of the inflated resected specimen revealed no evidence of cavity.

Pathologic Findings—The specimen measures 8 by 7 by 3 cm. Occasional oval and round necrotic foci are present which are chalky in appearance and measure from 1 to 2 mm in diameter. Bands of connective tissue traverse the intervening portions of the lung tissue.

Microscopically, the necrotic foci contain abundant calcium salts. They are surrounded by wide zones of hyalinized connective tissue. Other necrotic foci of a more recent nature show no calcium and are surrounded by granulation tissue.

Serial sections of the tissue show that the necrotic foci represent areas of tuberculous pneumonia which had undergone necrosis and in which calcium salts had been deposited in some of the older foci. No bronchi communicate with these foci. The serial sections do, however, reveal a minute cavity composed chiefly of hyalinized connective tissue. In some

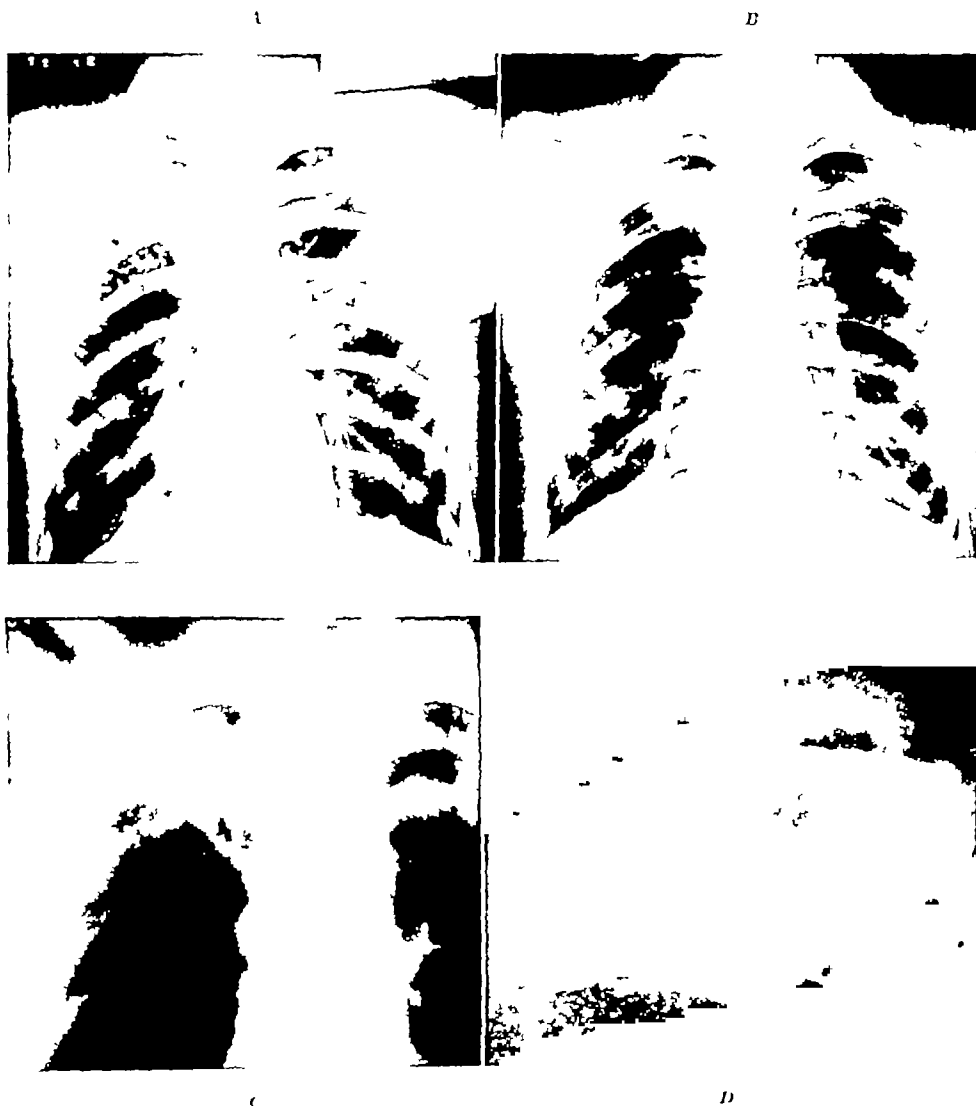


FIG. 2. *A, B*, Case 7. *A*, Ch. X-ray, Nov. 1, 1952. Streptomycin, 1.0 gm. daily, and PAS, 1.0 gm. daily, were begun Nov. 11, 1952. *B*, Chest X-ray, Aug. 1, 1957. After six months of daily streptomycin regimen was changed to twice weekly and PAS continued daily. All smears and cultures promptly became negative after starting streptomycin and PAS. *C*, Anteroposterior tomogram of right upper lung, June 25, 1957, prior to segmental resection on Aug. 1, 1957. *D*, X-ray of inflated resected specimen. Section of this showed inspissated cavity and 7 to 8 mm. x flow foci.



Fig. 1. E—Case 1. Photomicrograph of an inspissated cavity from which the necrotic material was removed for bacteriologic study. F—Photomicrograph of one of the residual areas of the cavity with a fragment of cartilage enclosed by necrotic material. G—Photomicrograph of one of smaller necrotic foci surrounded by a zone of foamy cells. Note the absence of specific tuberculous elements.

sections the presence of Langhans' giant cells and epithelioid cells speak for this cavity's tuberculous origin. Foreign body giant cells are also present in the wall. A bronchus opening into it is observed in some of the sections. There are no necrotic fragments within the lumen of the cavity or attached to its wall.

Bacteriologic Findings—A specimen containing chalky foci from this resected lung tissue showed no evidence of acid fast bacilli either on culture or smear.

CASE 7—G. G. was a 24 year old white man, with bacteriologically proved pulmonary tuberculosis since October, 1952 (Fig 2, A). Streptomycin, 1 Gm daily, and PAS, 12 Gm daily, were started in November, 1952, and continued without interruption until May 1, 1953, when the streptomycin regimen was changed to 1 Gm twice weekly. Sputum and gastric smears converted to negative promptly after initiation of chemotherapy and remained persistently negative from Jan 5 1953 until time of surgery. Six sputum and gastric specimens taken between May 15 and July 13, 1953, were negative on culture. X rays showed rapid resolution of the lesions on the left (Fig 2, B). By April, 1953, stability was established on the right without definite evidence of cavitation on tomograms (Fig 2, C). An apical and posterior segmental resection of the right upper lobe was performed on Aug 17, 1953 (Fig 2, D).

Pathologic Findings—The specimen measures 14 by 7.5 by 2.5 cm. There are occasional oval and round necrotic foci measuring from 3 to 5 mm in size. These are surrounded by a narrow gray capsule.

Microscopically, one of the larger necrotic foci (Fig 2, E) represents an inspissated cavity. Serial sections of this cavity show a fragment of cartilage enclosed by necrotic material lining the cavity wall (Fig 2, F). Further serial sections show the continuation of the same bronchus communicating with the cavity.

The other necrotic foci are surrounded by wide zones of hyalinized connective tissue with an almost complete loss of specific tuberculous elements (Fig 2, G).

Bacteriologic Findings—A single specimen consisting of pus from the inspissated cavity and several small necrotic foci was culture positive for *M. tuberculosis* although negative by smear.

CASE 8—R. C. was a 31 year old white man with bacteriologically proved pulmonary tuberculosis since April, 1952 (Fig 3, A). The patient received isoniazid, 150 mg daily, from June 12 to September 23, 1952, at which time streptomycin, 1 Gm twice weekly, and PAS, 12 Gm daily, were substituted for isoniazid. Chemotherapy was continued without interruption until the time of surgery. Sputum specimen taken Sept 3, 1952, showed 6 acid fast bacilli on smear, while a specimen taken Dec 1, 1952, was also positive on smear (Gaffky I) although negative on culture. All other specimens taken after May 23, 1952, including 10 specimens taken after Dec 1 1952, and prior to April 14, 1953, were negative by smear and culture. Roentgenograms indicated bilateral stability for six months prior to surgery, and a large coalescent infiltrate with irregular small annular densities in the shrunken right upper lobe at the time of surgery. A right upper lobectomy was performed on April 14, 1953.

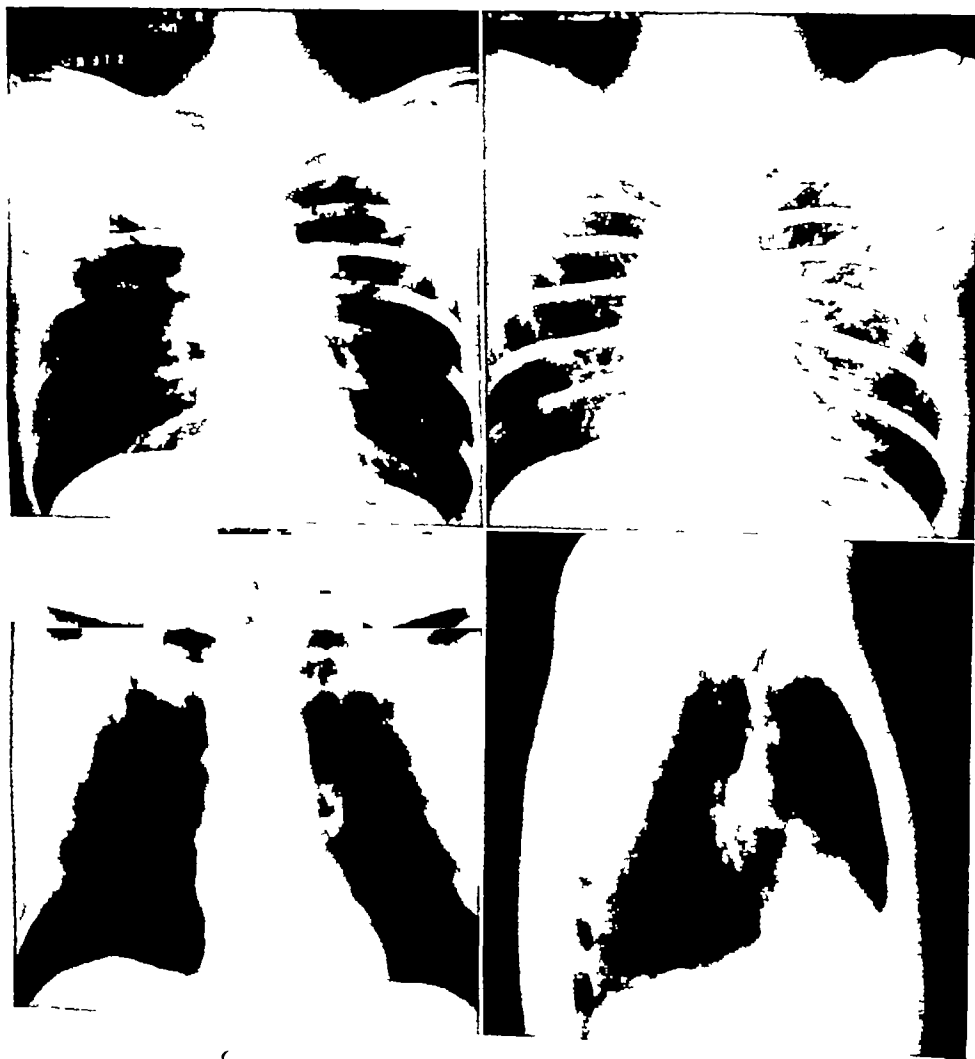
Pathologic Findings—The specimen of the right upper lobe measures 14.5 by 10.5 by 5.5 cm. A 4 by 3 by 2.5 cm filled in cavity is present in the superior portion of the lobe. The lumen is filled with firm inspissated yellow material which can easily be evacuated. A bronchus opens into the cavity and has a smooth lining. The cavity wall, which is gray in color, measures from 1 to 2 mm. Oval and round necrotic foci varying in size from 1 to 3 mm are present in the region around the cavity (Fig 3, E).

Microscopically, the lumen of the inspissated cavity is filled with fragmented necrotic particles containing abundant calcium and cholesterol. A narrow zone of tuberculous granulation tissue encloses the necrotic mass and is in turn surrounded by a thin layer of hyalinized connective tissue (Fig 3, F). A bronchus which opens into the cavity contains necrotic fragments in its lumen, similar to that which lies in the cavity (Fig 3, G). The small necrotic foci have an even appearance and are encircled by narrow hyalinized connective tissue.

Bacteriologic Findings—The necrotic contents of the right upper lobe inspissated cavity were positive for tubercle bacilli both on smear and culture. In like manner, smears of a portion of the unsloughed satellite necrotic lesions showed acid fast bacilli, while viable tubercle bacilli were recovered on culture.

A

B



C

D

FIG. 2. A, D—Case 8. A. Chest x ray, April 24, 1952. Isoniazid administered from June 12 to Sept. 2, 1952, and thereafter streptomycin and PAS. B. Chest x ray, April 10, 1953. The last positive sputum culture was obtained May 23, 1952, and the last positive smear Dec. 1, 1952. C. Anteroposterior tomogram, Feb. 4, 1953. D. Right lateral tomogram, March 4, 1953. The right upper lobe was resected April 14, 1953. The resected specimen showed a large cavity.

CASE 9—A. M., a 34-year-old white man, had had pulmonary tuberculosis since 1943. Therapy with streptomycin 1 Gm daily and isoniazid 150 mg daily, was initiated in September 1952, and was continued without interruption with the exception that the dosage of isoniazid was increased in December, 1952, to 300 mg daily and the strepto-

ministered only twice weekly. Sputum was positive by culture on July 23, 1952. Eleven sputum and gastric specimens taken between Dec. 19, 1952, and the time of resection were negative by smear and culture. X rays showed progressive clearing of the extensive bilateral disease in both upper lobes, with development of marked fibrosis and shrinkage and persistence of cavity in the right upper lobe (Fig 4, B and C). On May 18, 1953, a right upper lobectomy was performed. A widely patent cavity was demonstrated by X ray of the inflated resected lobe (Fig 4, D).

Pathologic Findings—The specimen of the right upper lobe measures 12 by 8 by 5 cm. A system of interlocking cavities is present in the posterior lateral aspect of the lobe just beneath the pleura and 2 cm from the apex. The cavity measures 3.5 cm in



Fig. 4. A, B—Chest X rays at time of discovery of pulmonary tuberculosis April 1952. Streptomycin and IAS dosage was begun June 21, 1952. C—Chest X ray April 17, 1953. Numerous smears and cultures between December 1952 and May 1953 were negative repeatedly. D—Anteroposterior tomographic view of right upper lobe cavity. E—X ray of inflated resected specimen May 18, 1953. Section of this showed clean-walled patent cavity with one small adherent necrotic focus.

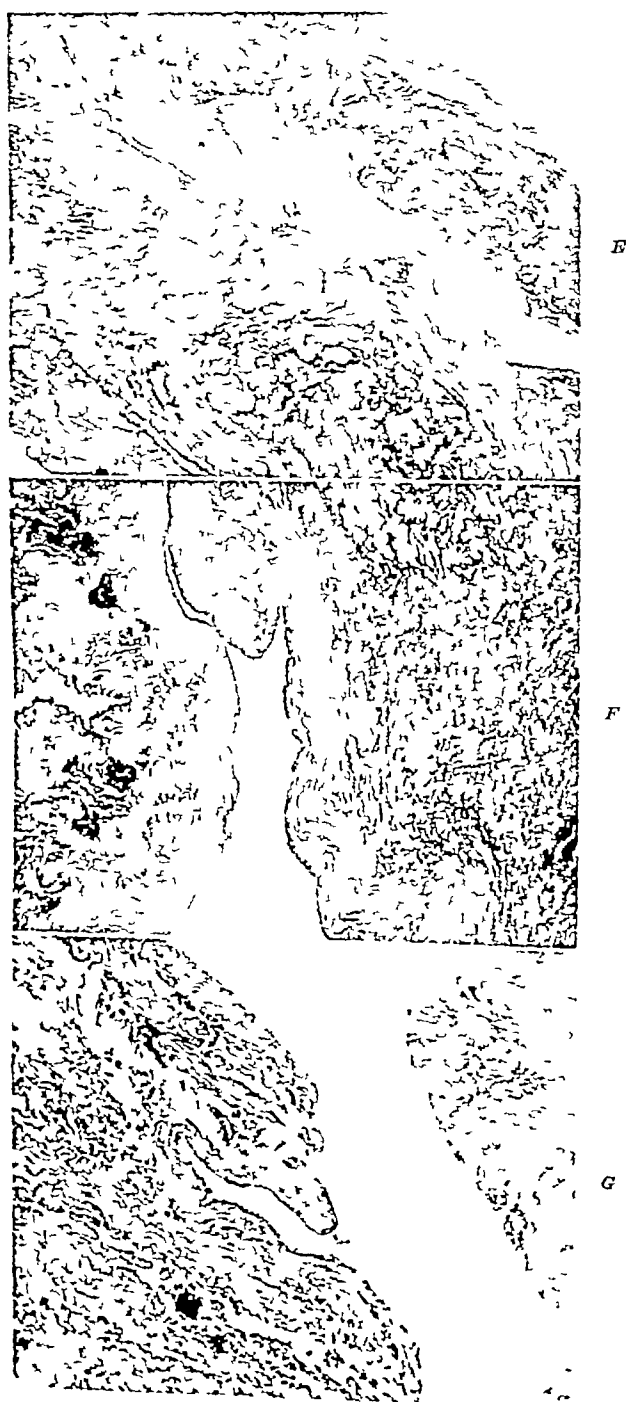


FIG. 4. E (—Case 11. E Photomicrograph of clean walled cavity and surrounding lung tissue. F Higher power view of same cavity wall showing hyalinized connective tissue without specific elements of tuberculois. G Photomicrograph showing bronchus opening into the same cavity.

the apicobasal direction, 2 cm in the mediolateral direction, and 2 cm in the anteroposterior direction. The cavity is lined by a smooth, shiny gray surface. A small, yellow necrotic fragment is attached to the wall in one small area. The cavity wall measures 1 mm in thickness. Near the cavity and in other portions of the lobe are 1 to 5 mm necrotic foci.

Microscopically, the cavity wall is composed of hyalinized connective tissue containing a few fibroblasts (Fig 4, *E* and *F*). Bronchial communications are present in several regions. The epithelium is intact up to its entrance into the cavity (Fig 4, *G*). The necrotic foci, which are often contiguously arranged, show the former structure of the lung parenchyma still discernible. These are surrounded by hyalinized connective tissue containing occasional epithelioid and giant cells.

Bacteriologic Findings—The wall and tissue surrounding a clean walled cavity was smear and culture positive for acid fast bacilli. In like manner, a clean walled cavity lying just inferior to and communicating with this previously mentioned cavity, and with a small necrotic zone, was positive on culture, although negative on smear. Two 4 mm puttylike foci in addition revealed acid fast bacilli both on smear and culture, as did a clump of small firm yellow foci.

Comment—Six of the 11 patients had received 4 to 8 months of chemotherapy, while 5 had received more than 8, although less than 12 months of chemotherapy immediately preoperatively. In all but one instance, streptomycin had been administered twice weekly, in combination with a second antimicrobial agent, during the period prior to surgery. Four, however, had received daily streptomycin for periods ranging from 2 weeks to 6 months prior to initiation of the twice weekly regimen.

It is of interest that in 3 of the 11 patients in this group, smears, but not cultures, were positive until one to two months prior to resection. The significance of positive smears, after cultures have become negative, to our knowledge has not been indicated to date.

Two of the patients in this group each had one isolated positive culture 4 to 6 weeks prior to resection, after having been culture-negative for a period of several months previously. It is of interest that no viable tubercle bacilli were found in the inspissated cavities of these individuals.

Also included in this group is an example of open healing under chemotherapy. This patient had had 11 months of preoperative chemotherapy and had been bacteriologically negative by smear and culture for more than five months preoperatively. It is significant that only one small necrotic fragment was attached to a smooth walled cavity which microscopically was composed of hyalinized connective tissue which had no specific changes for tuberculosis. The necrotic fragment and the cavity wall were smear- and culture-positive for acid fast bacilli. Patent communicating bronchi were demonstrated microscopically (Case 11).

CASE REPORTS—RE-TREATMENT CASES

Case 12—C. D., a 36 year old white man, had had pulmonary tuberculosis since February, 1948, and x-ray evidence of excavation in the left lung in 1952. Streptomycin, 1 Gm twice weekly in association with PAS, 12 Gm daily, was administered for a period of 90 days beginning Jan 12, 1952. In July, 1952, the patient was again started on streptomycin, 1 Gm twice weekly, with PAS, 12 Gm daily, and the regimen was continued without interruption until time of surgery. A single sputum specimen taken on Dec 31, 1952 was smear and culture negative, while a second specimen taken on Jan 2, 1953,

showed a Gaffky I on smear. Thereafter, six tracheal, gastric, and sputum specimens taken preoperatively were negative by smear and culture. The last positive culture had been obtained April 2, 1952. Serial films showed $\pm$ ray stability without definite evidence of cavity for 6 months prior to resection. A left upper lobectomy and segmental resection of the superior segment of the left lower lobe was performed on April 28, 1953.

Pathologic Findings.—The specimen of the left upper lobe and the dorsal segment of the left lower lobe measures 18 by 11 by 4 cm. Necrotic foci are present in both lobes. In the upper lobe they vary in size from 2 mm. to 1.5 cm. while in the lower lobe they are from 3 mm. to 2 cm. in size. The smaller foci are firm and flat while the larger ones, which represent filled in cavities, contain soft yellow material which is easily evacuated. The lower lobe cavity shows softer material than the upper lobe cavities. A branch of the lower lobe bronchus communicates widely with the cavity.

Microscopically, the filled in cavities of both lobes contain fragmented necrotic fragments. Those of the lower lobe contain in addition many neutrophils (liquefaction). Those of the upper lobe contain cholesterol and calcium deposits. The cavity walls are thin showing tuberculous granulation tissue with an abundance of collagen fibrils. The smaller necrotic foci show a similar wide capsule of tuberculous granulation tissue.

Bacteriologic Findings.—The central necrotic portion of a 5 mm. inspissated cavity was positive for acid fast bacilli both on smear and culture while the wall of this cavity was positive on culture although negative on prolonged examination of smears. The wall and central necrotic component of a second inspissated cavity were pooled for bacteriologic study and again showed acid fast bacilli both on smear and culture. Viable tubercle bacilli likewise were recovered from two small (pooled) necrotic foci.

CASE 13.—W. A. was a 31 year old Negro man with bacteriologically proved bilateral pulmonary tuberculosis since 1949 and bilateral spread of disease plus evidence of a small cavity in the right upper lobe under a thoracoplasty in 1952. Prior to 1951 and in January, 1952, the patient received a total of 320 Gm. of streptomycin in discontinuous courses administered daily but without regular PAS therapy. Complete resistance to streptomycin was observed, beginning May 1951. The patient was placed on isoniazid 300 mg. daily and streptomycin, 1 Gm. twice weekly on Nov. 7, 1952, and was continued on this regimen until the time of surgery. Four sputum specimens taken after Nov. 5, 1952 were positive on smear but negative on culture. A single isolated specimen taken Dec. 8, 1952, was culture positive. Bacilli isolated on this occasion were resistant to 10 μ g PAS but susceptible to 0.2 μ g isoniazid and 3.5 μ g streptomycin per milliliter of medium. Six sputum and gastric specimens taken subsequent to Dec. 8, 1952 and prior to surgery were negative by smear and culture. X rays showed progressive clearing of the infiltrative lesions in the right lower and the left lung field and were considered stable by March 1953. On June 17, 1953, a right upper lobectomy plus additional thoracoplasty was performed. At the time of resection the small cavity under the thoracoplasty could no longer be seen on tomograms or on x ray of the inflated resected lobe.

Pathologic Findings.—The specimen of the right upper lobe measures 11 by 6 by 3 cm. Occasional necrotic foci varying in size from 1 to 3 mm. are present in the lung tissue and are surrounded by wide connective tissue capsules. There is a great increase of connective tissue within the lung parenchyma. There is no evidence of the presence of an inspissated cavity.

At the time the specimen was first opened for bacteriologic examination a small smooth walled cavity was observed. Approximately one half of it was removed for bacteriologic studies. After the specimen was fixed in formalin the remaining portion of the cavity was not seen.

Microscopically, large portions of the lung tissue are replaced by loose and dense connective tissue. Lying within the zones of fibrosis are occasional small necrotic foci which are encircled either by hyalinized connective tissue or granulation tissue showing an abundance of collagen fibrils. A few small similar foci are present which contain no necrotic centers.

Serial sections reveal a small cavity space lined by a zone of hyalinized connective tissue. There are no specific tuberculous elements present within it.

Bacteriologic Findings—Half a clean walled cavity, including the wall and lung parenchyma, was cultured and was positive for acid fast bacilli on culture although negative on smear.

CASE 14—L. R. a 34 year old white man, had had pulmonary tuberculosis since 1949. Between August, 1949, and February, 1950, the patient received an initial 60 day course of streptomycin, 1 Gm daily, followed by a subsequent 15 day course of streptomycin, 1 Gm daily, plus PAS, 12 Gm daily. This in turn was followed by PAS alone for approximately 3 months. On Nov. 22, 1952, treatment with isoniazid, 300 mg daily, and PAS, 12 Gm daily, was initiated, this regimen was continued to the time of surgery. On Nov. 17, 1952, sputum was positive by smear and culture for acid fast bacilli. Nine sputum and gastric specimens taken between Dec. 3, 1952, and April 13, 1953, were negative by smear and by culture. In November, 1952, the patient revealed a left apical infiltrate with out distinct evidence of excavation. Lateral tomography, however, disclosed a thick walled 2.5 cm cavity in the superior segment of the left lower lobe. A repeat left lateral tomogram taken April 21, 1953, revealed no evidence of a patent cavity. A resection of the dorsal segment of the left lower lobe was performed on May 4, 1953. A ray of the inflated resected specimen revealed an inspissated cavity measuring 1.5 by 1 cm in size.

Pathologic Findings—The specimen measures 10 by 9.5 by 2.5 cm. A filled in cavity, measuring 1 by 1 cm by 8 mm, is present. The lumen is filled with soft, yellow material. The cavity wall measures 1 mm in thickness. A bronchus cannot be traced into the cavity with any definiteness. Occasional firm necrotic foci varying in size from 2 to 5 mm, are present in the area around the inspissated cavity.

Microscopically the necrotic contents of the cavity are fragmented and intermingled with mucin and neutrophils. The cavity wall is composed of granulation tissue with an abundance of collagen fibrils. A bronchus opens into the cavity and shows an intact epithelial lining up to its entrance into the cavity. The small necrotic foci show the ghost forms of the underlying lung structure. They are surrounded by hyalinized connective tissue containing a few fibroblasts and epithelioid cells.

Bacteriologic Findings—The central necrotic component of an inspissated cavity was positive for acid fast bacilli both on smear and on culture while the wall of this cavity was negative for tubercle bacilli.

CASE 15—L. B. a 26 year old Negro man, had bacteriologically proved pulmonary tuberculosis and right upper lobe cavitation. In October, 1951, the patient was started on streptomycin, 1 Gm daily, and PAS, 12 Gm daily, for a period of 4 months, this regimen was continued without interruption with the exception that the streptomycin was administered only twice weekly after the first 6 weeks. On Nov. 7, 1952, isoniazid therapy was started and continued without interruption until the time of surgery. PAS, 12 Gm daily, was added to the regimen on Nov. 19, 1952. Ten of 11 specimens taken between Nov. 20, 1952, and May 6, 1953, were negative on culture. A single specimen taken Dec. 8, 1952 was positive for tubercle bacilli. Smears were positive on 6 of these specimens, all 5 specimens taken after Jan. 5, 1953, being negative on smear as well as on culture. X rays showed progressive shrinkage of the lesion in the right upper lobe, as well as clearing of the seeding in the left lung. Stability on X ray was obtained by February, 1953. At that time the right upper lobe was converted into a shrunken atelectatic mass with the cavity lost to view. A right upper lobectomy was performed on May 19, 1953. X ray of the inflated resected lobe revealed no cavity.

Pathologic Findings—The specimen of the right upper lobe measures 7 by 6 by 4 cm. A radial scar measuring 3.5 by 1 cm by 7 mm is present in the posterior and lateral aspect of the lobe. A bronchus ends abruptly in the scar. Superior to the scar is a 4 mm soft liquefied focus surrounded by a narrow gray zone. A group of necrotic foci, varying in size from 1 to 3 mm, are also present in other parts of the lung tissue.

Microscopically, the radial scar is composed of loose and dense connective tissue which gradually tapers off among the surrounding alveolar septa. The liquefied focus shows the necrotic fragments intermingled with neutrophils. This is encircled by a layer composed of collagen fibrils with epithelioid and giant cells.

Serial sections show a bronchus opening into the cavity (the 4 mm liquefied focus described above). The epithelial lining is intact as it enters the cavity.

Bacteriologic Findings—A piece of lung tissue, containing small liquefied areas, was snapped for bacteriologic study although smears were positive for acid fast bacilli organisms were not recovered on culture. A bunch of small necrotic partially calcified foci in contrast were positive for tubercle bacilli on culture although negative by smear.

Comment—Four of the 19 patients had received previous courses of antimicrobial therapy, terminating 3 to 21 months prior to institution of the preoperative course of chemotherapy. All had received from 5 to 10 months or more of chemotherapy immediately preoperatively and all were bacteriologically negative by smear and culture for at least 3 to 6 months preoperatively. Cavity closure was observed morphologically in each case.

Control Cases—Four of the 19 cases were considered as controls. Of these, two (Cases 16 and 17) had received either no or essentially no chemotherapy preoperatively while two had open cavities (Cases 18 and 19) at the time of resection. One of the two patients with open cavities had had 12 months of preoperative chemotherapy (Case 18), while cultures (but not smears) from this patient had been negative for eight and one-half months prior to surgery. All lesions from all control patients were culture-positive for *M. tuberculosis*.

DISCUSSION

As indicated by data reported herein and also in a previous publication by the present authors²⁸ viable tubercle bacilli were recovered from 7 of 11 original treatment cases and from all of 4 retreatment and 4 control cases. Such viable cells were detected as frequently from original as from retreatment cases, and as frequently from patients who had had 8 to 12 months as from those who had had 4 to 8 months of chemotherapy.

Most of the patients in this group received streptomycin twice weekly in combination with PAS daily. Five patients received isoniazid daily together with streptomycin or PAS. Since none of the patients in our series received daily streptomycin during the immediate preoperative period no comparison can be made therefore between daily and twice-weekly streptomycin regimens. Indeed the data to date fail to suggest any significant difference among the regimens used in this series.

The majority of patients in the group reported by D'Esopo and his associates¹ received daily streptomycin in combination with PAS. D'Esopo²⁹ stated that this may be one of the factors which may account for the difference between their findings and ours. He pointed out that patients receiving streptomycin 1 Gm twice weekly, and PAS, 12 Gm daily, show an appreciably higher incidence of viable bacilli than those receiving streptomycin daily. He found the incidence of viability in patients receiving twice-weekly streptomycin to be approximately 35 per cent. The incidence in our study on similar regimens is 73 per cent. He also indicated that as far as preoperative chemotherapy is concerned there is a greater opportunity for bacteriologically negative results if it is used in excess of 8 and possibly in excess of 12 months. The statement has been made by D'Esopo and associates, however, that the patients whom they have selected for resection were mostly ones who had been treated "with an arbitrary minimum period of four months of preopera-

tive chemotherapy" An attempt was made in the selection of our material for this study to follow as closely as possible the criteria set down by these authors. Thus, all patients in this report received more than 4 months of chemotherapy, a number received more than 8 months, although none received more than 12 months. The findings of Steele and his associates⁷ are of interest in this respect. Their results show no great difference in the recovery of viable bacilli in the two regimens. In Steele's group receiving original treatment for 4 to 8 months, viable organisms were recovered in one of 16 cases receiving daily streptomycin and in none of 6 cases receiving the drug twice weekly. In the group receiving daily therapy from 8 to 24 months, viable bacilli were recovered from 3 of 33 cases and from 3 of 39 cases receiving streptomycin twice weekly.

If the difference in the viability of organisms lies in the use of daily streptomycin over long periods of time, then this would greatly alter the approach toward chemotherapy. The almost universally accepted use of streptomycin today is on a twice weekly basis combined either with PAS or isoniazid daily.

The recovery of viable strains of *M. tuberculosis* from the lesions described in the present report leads one to wonder what may be their clinical significance. It is of interest in this regard that, on subculture, all strains were highly virulent for guinea pigs. The significance of guinea pig virulence in relation to the ability of an organism to produce disease in man has never been firmly established, nonetheless, it has been assumed implicitly over the years. Of further interest is the fact that all strains recovered were highly susceptible to 0.1 μ g of isoniazid per milliliter of medium and all but one was susceptible to 1 μ g of streptomycin per milliliter of medium.

From the data presented, it seems probable that improved cultural techniques may increase the frequency with which viable tubercle bacilli may be recovered from necrotic foci, including inspissated cavities. These findings, however, suggest further that either duration of chemotherapy may be more important than has been indicated previously and must exceed 12 months in order to assure sterilization of the lesion or the chemotherapeutic regimen employed may be more important than has been apparent in the past.

Of prime importance in relation to the present study is the fact that tubercle bacilli may survive in inspissated tuberculous cavities even after extensive chemotherapy and that the survival of these organisms can be demonstrated in so large a proportion of cases.

Tuberculous cavities which close by inspissation of the necrotic contents, under chemotherapy show a communicating bronchus. As has already been pointed out the bronchus is reepithelized as it enters the cavity. The reepithelized surface of the bronchus at the bronchocavitary junction means that there will be a communication with the necrotic contents of the cavity and other parts of the lung parenchyma. Since the necrotic contents may again become liquefied through the invasion of polymorphonuclear leukocytes the presence of viable organisms remains as a threat to the patient.

There are several features of the fate of the inspissated cavity which still need explanation. There are many cases where cavities have closed with in

spissation of the necrotic contents under chemotherapy which have been observed for several years after the cessation of such therapy. No spread of disease has been observed in such cases. Other similar cases have been followed where the inspissated cavity has shelled out (during chemotherapy and also subsequent to it) without the development of new tuberculous disease in other parts of the lung.

We believe that Case 11 is of extreme significance in our approach to the evaluation of open healing. Viable bacilli were found in a single necrotic fragment attached to a cavity wall. Microscopic examination of the cavity wall in various sections showed a loss of specific tuberculous elements. Perhaps serial sections of this cavity may have shown some tuberculous foci still present. The observations in this case pose several questions. Patients showing open cavity healing under prolonged chemotherapy with long periods of negativity may still contain necrotic fragments in which are lodged viable tubercle bacilli. Since there is no method of determining roentgenographically when all the necrotic fragments have disappeared from the lumen of the cavity and since all investigators have found a high percentage of positive cultures from resected cavities despite negative sputum and gastric cultures, it is at present agreed that chemotherapy should not be stopped until all open cavities are resected.

SUMMARY

A clinicopathologic correlation with microbiologic observations was performed on resected tuberculous lesions from 19 patients. Four of the 19 patients were considered as controls. Fourteen of the remainder had received from 4 to 12 months of chemotherapy preoperatively and showed morphologic evidence of cavity closure. One had received 11 months of chemotherapy preoperatively and showed evidence of an open cavity with healing. The recovery of viable tubercle bacilli from the 4 control patients and also from 11 of the 15 original and retreatment patients has been reported.

No correlation between the presence of viable tubercle bacilli in tuberculous lesions and duration of chemotherapy, drug regimens used, duration of the preoperative period, noninfectiousness, or the anatomic nature of the lesions could be demonstrated in this study.

Based on the findings reported herein, inspissated cavities with communicating bronchi after prolonged chemotherapy on regimens universally used today are potentially dangerous in a high percentage of cases.

We should like to express our thanks to Mr. Masay M. Nakamura, for the illustrations in this article.

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DISCUSSION

DR ALFRED GOLDMAN, Beverly Hills, Calif.—About two or three years ago we began to study tubercle bacilli from resected surgical specimens. At that time we were interested in streptomycin resistance in streptomycin treated patients who had lung resection for tuberculosis. Approximately 120 surgical specimens were cultured from the resected lungs and we could never determine in cultures from any of these specimens in which we recovered tubercle bacilli any difference in the resistance to streptomycin of the organisms recovered and the resistance of organisms found in the sputum. Subsequently we studied another group of 41 patients who had segmental resections. In this group, the data on which are published in the last Transactions of this Association, we recovered only two positive cultures in those patients of whom there were 26, who had negative preoperative sputum. Smears and cultures were negative for at least six months preoperatively. Subsequent to this, we have a third group of patients now under study, and it is with regard to these that I wish to speak.

This is a group of 29 cases none of which have had less than four months of treatment, isoniazid, PAS, and streptomycin, 1 Gm, twice a week, whereas the original group we studied had streptomycin daily five times a week. In this group we have been able to recover viable tubercle bacilli with more assiduous study than the routine study used previously, approximately thus far (and the study is still going on) 50 per cent as compared with the 7 per cent just reported of viable tubercle bacilli in these lesions, most of which were closed lesions or blocked cavities.

(Slide) In this respect our results parallel those which were just reported by Dr Auerbach. This slide represents a segmental resection in a patient who had a filled in cavity clinically, and this is the drawing of the segmental resection preoperatively the sputum was negative and the culture from this is negative.

(Slide) This is a summary of the present status of this study. We may for practical purposes conclude that all the cases below this line had negative preoperative sputa on smear and culture. Most of these were negative on more than twelve cultures. Yet you see that about five of them had positive viable tubercle bacilli obtained from the specimens.

To me this type of study has a great significance in the management of the patient, and this is the point I wish to make. Today it seems that many of our beds are taken up by patients in the sanatoria who are no longer a menace to society. We can use these bacteriologic criteria obtained from our surgical specimens for decision as to early ambulation and early discharge. For example, take the patient whose specimen you saw projected in the slide, that patient was ambulatory within six weeks after segmental resection and was discharged from the hospital within four months. It seems to me that this type of study offers the opportunity for us to acquire more scientific criteria as a basis on which to make the decision of early ambulation and early discharge after pulmonary resection.

DR EDWIN T. GRACE, Brooklyn, N. Y.—When we are speaking of chemotherapy I think it is important to consider certain fundamentals in medical semantics and to define just what we mean by modern chemotherapy. We have some very reliable data on this bacteriologic phenomenon. First these specimens of viable tubercle bacilli from the so called healed lesions are probably bacteriologic mutants that one would anticipate emerging from inadequate topical chemotherapy. With modern chemotherapy it is necessary to go back almost 100 years and integrate the Mendelian law (genetics) directly to current clinical bacteriology when these laws are applied the viable bacteria are probably mutant strains arising from the original tubercle bacillus. Second if we carefully study the original Petri dish reported by Fleming twenty five years ago most of the bacteria present about the penicillin were destroyed and the lethal effect increased in direct proportion to its intimacy with the chemotherapeutic agent (penicillin) and the few remaining colonies in this same area we now recognize as resistant strains or mutants. Third Florence, shortly after World War II spoke at Yale University and clinically brought out the important point of applying the chemotherapeutic agent directly to the wound. In a case report from the North African campaign of the British Army, he applied topically not more than 20,000 units of penicillin for two days into a ghastly wound of the thigh in which all the muscle were split across by a shell and the sepsis was cleared up.

(Slide) If we use this therapeutic concept, we now recognize that the most lethal chemotherapeutic combination to destroy the tubercle bacillus and prevent the emergence of resistant strains is a combination of streptomycin and isoniazid (Streptohydrazid) applied topically with a detergent in an aerosol mist to the primary focus in the lung. This x ray shows a moderately advanced lesion in the apex of the left lung of two months' duration with a combination of 236 mg of isoniazid and 1 Gm streptomycin dissolved in 8 cc of a detergent (Alevaire) inhaled deeply in an aerosol mist for three weeks and a like amount given intramuscularly with PAS and isoniazid (Parasal) orally. In three weeks the lesion had cleared, and sputa were negative in four weeks. Eleven month follow up showed no symptoms, all six cultures negative, chest and tomograph negative. He has been gainfully employed for six months as a dancing teacher. In other words, the important thing we must realize at the start of all chemotherapy is to direct our therapy not to induce further chronicity by allowing preventable bacteriologic mutants (viable tubercle) to emerge.

(Slide) At the meeting held after the war in St. Louis in 1947, I noted that, from declassified data obtained from the technical division of chemical warfare on so called gas warfare, it was possible to use an antibiotic (penicillin) with a detergent (Zephiran chloride) to produce a mist and inhale it deeply into the lung for therapeutic purposes. At that meeting I had this slide with me but hesitated to present it because of the limited follow up of only a few months. However, I did show it later to some members of this Association from Houston, Texas. This patient, after two years of unsuccessful treatment, had a severe hemorrhage. She was given fundamentally the same type of inhalation therapy for a longer period, one month, using penicillin and streptomycin dissolved in a detergent solvent. In four months the extensive cavity of the left lobe, the infiltration of the right had become fibrosed and all cultures (11) are negative. With semiannual checkups for seven years this patient has been an active housewife and is in good health.

The clinical importance of this paper of Hobby, Auerbach, and associates from our experience is that with chemotherapeutic agents now available to prevent, probably, the viable tubercle bacilli or mutants from emerging this simple but biologically sound concept should be considered before any surgical or compression therapy.

[Dr. Grace asked the editor for permission to include in his discussion a letter from Dr. Vernon Bryson a geneticist, concerning the interpretation of Fleming's original penicillin plate—Editor.]

Dear Dr. Grace

You will recall that we recently had a conversation on the nature of the colonies evident in your photograph of Dr. Fleming's original discovery of penicillin. It seems to me most likely that what we observe here is inhibition, rather than the selection of mutants. This viewpoint is supported by the visual evidence that colonies are well separated and growing normally on the Petri dish in regions well removed from the contaminating penicillin. If my recollection is correct, there could have been no more than about fifty bacteria on the plate (assuming a random distribution) at the time the colonies started to grow. This number is too small to include rare types of mutants except on very infrequent occasions arising from early mutation in the growth of the parent culture. As a second point all colonies appear to be identical in their response to the antibiotic, growing if distant and failing to grow if close to the penicillin. A single resistant mutant would grow well and stand out by contrast with its inhibited neighbor, but no such conspicuously resistant colony can be found. All seem inhibited about equally. One may therefore assume a small number of sensitive cells were present together with the contaminant and that the size eventually reached by colonies growing from these cells was controlled by the distance from the penicillin source.

Yours sincerely,
VERNON BRYSON

DR EDWARD S WELLES, Saranac Lake, N Y—I am very much encouraged by Dr Auerbach's paper. I have been one of the most ardent disbelievers in the death of the tubercle bacillus. At an open meeting recently, Mr Steenken assured us that the organisms were dead and, at the same panel table, I assured the audience that they must not believe a word of it. His low percentage of cultures in animals lent a good deal to his side of the argument. Now that Dr Auerbach has told us about 73 per cent positives, I feel that if he goes on with his present methods or possibly finds an even better method, his 73 per cent will rise to 98 per cent. I hope that a year from now Dr Auerbach will come to Atlantic City and show us a much higher percentage than even 73 per cent.

I should like to ask if this experiment is being carried on with another group of cases that have had streptomycin therapy daily.

DR OSCAR AUERBACH, East Orange, N J (Closing)—In answer to Dr Welles' question I should like to say that beginning last month we have started a joint study with Dr Desopo and his group at West Haven in which he is submitting his material to us at the beginning of each month, and we at East Orange are processing our material at the end of each month, and we hope in that way to be able to answer one or both of the questions brought up in our discussion.

There are one or two things I would like to say about this study. One is that this basically is a research problem, the routine laboratory does not have the facilities to carry on this extensive bacteriologic study. Another point of interest is that those cases in which we can find no communicating bronchus at the time of gross examination are submitted for serial sections, another procedure which cannot be done in the routine laboratory. I should like to reiterate at this time that when we made the statement that the necrotic foci which have never liquefied do not communicate with the bronchus, this has been ascertained by serial section. We do not consider the small necrotic foci potentially dangerous on the basis of these serial studies. That the process is open to many questions is illustrated by the fact that one of the cases gave a positive sputum by smear and culture shortly before section. We saw nothing on gross examination except small necrotic foci and when we proceeded with serial section of the lung we found a partially epithelialized cavity with a few Langhans giant cells still present in the wall without any necrotic fragments present. Thus the one necrotic fragment still present was evacuated preoperatively and we were able to get a positive smear and culture from it.

These are all very intriguing and almost hopeless problems from the point of view of roentgenographic and clinical studies which we hope will be cleared up in the future.

RESECTION SURGERY IN TUBERCULOSIS

COMPLICATIONS AND AFTER-HISTORY

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AT THE present time, resection surgery in pulmonary tuberculosis is in the phase of development and study. Ryan, Medlar, and Welles¹ have proposed the thesis based on extensive pathologic investigation, that it is feasible to excise residual necrotic lesions which constitute a threat to the continued health of the tuberculous patient. Chamberlain and associates² have reviewed those surgical principles which are applicable to segmental resection, including the postoperative care. Our purpose is to present a series of pulmonary resections which were performed pursuant to the thesis advanced by Ryan, Medlar, and Welles and in accord with the surgical principles described by Chamberlain and associates. A review will be made of the type of operation, the attendant risks, the postoperative complications, and the initial period of the "after history." These data will in due course, permit comparison with results obtained by other regimens and will aid in determining the proper use of resection procedures.

The present study includes 589 consecutive pulmonary excisions for tuberculosis performed during the years 1949-1953 inclusive, 246 at the Mt. Morris Tuberculosis Hospital and 343 at the Hermann M. Biggs Memorial Hospital. Not included are 10 additional patients whose chests were opened surgically, but in whom no resection was performed because the disease was too wide spread in 7, no nodular disease was found in 1, and a change in the patient's condition did not permit resection in 2. One of the latter had cardiac arrest treated by thoracotomy and massage with complete recovery. All the patients received drug therapy utilizing streptomycin, para-aminosalicylic acid, isoniazid or viomycin in one or more combinations. The duration of this therapy varied rather widely. In general operation was undertaken when it was believed that maximum x-ray clearing of disease had occurred, but no arbitrary period of stability was required. Absence of tubercle bacilli from concentrated sputum was considered highly desirable but not an absolute criterion for acceptance for surgery. An adequate respiratory reserve was a prerequisite and function studies including bronchspirometry, were made as indicated.

The patients have been divided into two subgroups. (1) The "Salvage" group totaling 110 included 55 patients with thoracoplasty failure, 31 with cavities exceeding 1 cm in diameter persisting under pneumothorax or despite chemotherapy, 8 with destroyed lungs, 7 with tuberculous empyemata, and 9 with bronchostenosis. For these 120 operations were performed, and (2) the

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Hermann M. Biggs Memorial Hospital
Mt. Morris Tuberculosis Hospital

"Elective" group, totaling 397, had 469 operations. The indications for operation in this group were the presumed presence of necrotic lesions which had persisted after a course of drug therapy and the probability that these lesions continued to harbor viable mycobacteria. The hazard of relapse was thought to be more serious than the risk of operation.

OPERATIONS COMPLICATIONS—MORTALITY

We will review a total of 589 operations, 74 of which were on the contralateral lung while 8 were second operations on the same side. The operations are identified by the largest unit of lung removed; lesser accessory procedures being omitted. That is a lobectomy with an associated subsegmental resection is recorded as a lobectomy. The stage of disease (N.T.A. Standards, 1950) here presented reflects the extent at the time preoperative drug therapy was initiated; changes resulting from drug therapy being disregarded.

TABLE I OPERATIVE PROCEDURES BY STAGE OF DISEASE PRIOR TO DRUG THERAPY

PROCEDURE	STAGE PRIOR TO DRUG THERAPY			
	TOTAL	MINIMAL	MODERATELY ADVANCED	FAR ADVANCED
All procedures	589	114	354	121
Pneumonectomy	40	—	10	30
Lobectomy	129	5	82	42
Segmental resection	214	38	149	27
Subsegmental resection	206	71	113	22

TABLE II NUMBER AND TYPE OF OPERATIVE PROCEDURES BY YEAR AND SUBGROUP

YEAR OF OPERATION	TOTAL PROCEDURES		PNEUMONECTOMY		LOBECTOMY		SEGMENTAL RESECTION		SUBSEGMENTAL RESECTION	
	SAL VAGE	ELEC TIVE	SAL VAGE	ELEC TIVE	SAL VAGE	ELEC TIVE	SAL VAGE	ELEC TIVE	SAL VAGE	ELEC TIVE
Total	120	469	36	4	57	70	18	198	9	197
1949	18	4	6	—	9	2	3	1	—	1
1950	22	16	8	1	12	6	1	7	1	2
1951	28	63	10	1	13	12	3	25	2	25
1952	15	201	2	1	6	20	4	71	3	109
1953	37	185	10	1	17	30	7	94	3	60

TABLE III CAUSES OF DEATH POSTOPERATIVE AND LATER

	SALVAGE	ELECTIVE
<i>Deaths—early postoperative</i>		
Cardiorespiratory failure	6*	—
Serious blood loss	0	2
Vascular accident	1	1
Intrapulmonary bleeding	1	1
Cardiac arrest	1*	1
Progressive tuberculous (spread)	2*	—
<i>Deaths after 60 days</i>		
Tuberculous—contralateral at 27 mo	1	—
Amyloid disease at 40 mo	1	—
Unexplained hemoptysis at 5½ mo	1	—

* Value does not contribute to one in each group.

Table I presents the type of operation by stage of disease and is self-explanatory. Fig. 1 shows the distribution of operations graphically.

Table II presents by calendar year the number of elective and salvage operations performed and reflects a change in the choice of therapy, based in part upon the observed safety of the procedures used

Deaths—Table III shows the 19 deaths, all but 3 of which occurred within sixty days after surgery. Fourteen of the early deaths were directly related to the operation while 2 were due to progressive tuberculosis following a post operative spread of disease. Of the late deaths, 1 was due to an exacerbation of tuberculosis, 1 to amyloid disease, and a third to an unexplained massive pulmonary hemorrhage occurring six months after operation.

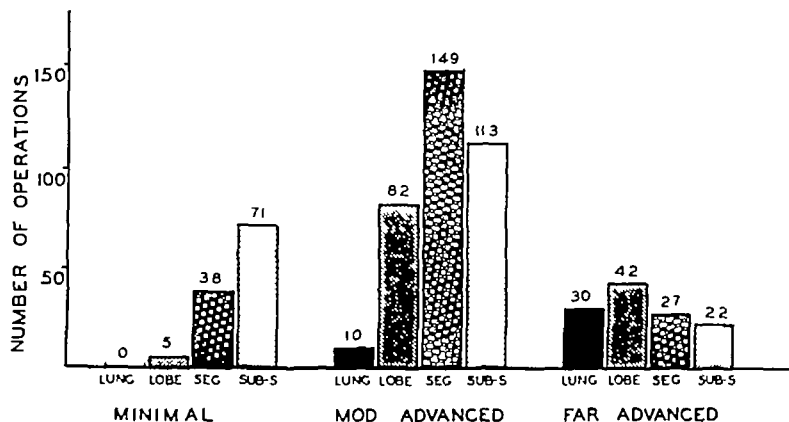


Fig 1—Pulmonary resections for tuberculosis by portion of lung removed and stage of disease

Table IV shows the deaths in relation to the type of operation

Complications—The frequency and severity of nonfatal complications is particularly important in assessing the use of surgery in “elective” cases. Seventy-nine or two thirds of the 120 salvage operations and 387 or four fifths of the 469 elective procedures were uncomplicated.

TABLE IV DEATHS TO SHOW RISK, AND TIME OF DEATH IN RELATION TO OPERATIVE PROCEDURE USED

PROCEDURE	TOTAL PROCEDURES			NUMBER OF DEATHS	
	SALVAGE	ELECTIVE		SALVAGE	ELECTIVE
Pneumonectomy	36	4	Postoperative	6	—
			Late	1	—
Lobectomy	57	70	Postoperative	5	1
			Late	1	—
Segmental resection	18	198	Postoperative	—	2
			Late	1	—
Subsegmental resection	9	197	Postoperative	—	2
			Late	—	—

Table V shows that serious complications were significantly more frequent among the salvage operations than among the elective operations and more frequent among lobectomies and pneumonectomies than among the less extensive excisions.

TABLE V COMPLICATIONS BY OPERATION, SALVAGE OR ELECTIVE. IN THE FEW INSTANCES OF DOUBLE COMPLICATIONS FOR ONE OPERATION, ONLY THE MOST SERIOUS IS LISTED

	TOTAL		LUNG		LOBE		SEGMENT		SUBSEG	
	S	E	S	E	S	E	S	E	S	E
TOTAL NUMBER OF PROCEDURES	120	469	36	4	57	70	18	198	9	197
SERIOUS COMPLICATIONS AMONG SURVIVORS										
Vascular accidents	-	3	-	1	-	-	-	2	-	-
Tuberculous empyema with bronchopleural fistula	3	1	-	-	3	1	-	-	-	-
Nontuberculous empyema with bronchopleural fistula	-	4	-	-	-	-	-	4	-	-
Nontuberculous empyema without bronchopleural fistula	1	3	1	-	-	1	-	2	-	-
Tuberculosis of bronchial stump	2	-	-	-	1	-	1	-	-	-
Tuberculous spread (within 2 mo of operation)	5	-	2	-	2	-	1	-	-	-
Late tuberculous exacerbations	2	3	1	-	1	-	-	-	-	3
Nontuberculous pneumonia	-	2	-	-	-	-	-	1	-	1
Total serious complications	13	16	4	1	7	2	2	9	-	4
MINOR COMPLICATIONS AMONG SURVIVORS										
Late alveolar leaks	1	29	-	-	-	1	-	20	1	8
Persisting pneumothorax pocket	-	12	-	-	-	1	-	8	-	3
Hemothorax	-	12	-	-	-	1	-	7	-	4
Persistent pleural fluid (uninfected)	-	5	-	-	1	-	-	4	-	-
Bronchial secretions requiring aspiration	6	5	-	-	5	3	1	2	-	-
Chest wall infection	2	2	1	-	1	-	-	-	-	2
Miscellaneous minor complications	-	2	-	-	-	-	-	2	-	-
Total minor complications	9	67	1	-	7	6	1	43	1	17

It is convenient to discuss the complications in relation to the site of occurrence pulmonary vascular system, pleural space, and bronchial tree

Vascular accidents The 8 vascular accidents occurring during surgery were relatively more frequent among the salvage cases. Because of bleeding from torn vessels 2 planned segmental resections and 2 planned lobectomies were converted to pneumonectomies, 1 of the patients in the latter group died. In 2 of the removed lungs, the pathologist noted unusually thin pulmonary artery walls with associated delicate lung structure. In a fifth case a torn left pulmonary artery was closed by sutures at the pericardium, resection was not performed, but cyanosis persisted to death hours later. Permission for autopsy was not obtained. A torn left pulmonary artery during lobectomy of a residual lower lobe was controlled but the patient died due to excessive volume of blood loss. In 1 case bleeding was a factor in cardiac arrest. In the eighth case the ligatures slipped from the left pulmonary artery just before completion of a pneumonectomy. The bleeding was controlled and the patient recovered.

Pleural space complications Chamberlain and co-workers have emphasized the necessity for evacuating air and blood from the pleural space and securing prompt re-expansion of the lung. The two elements of this problem are reviewed separately.

(a) Air enters the pleural space from the raw lung surfaces which are present following resections with the exception of a few lobectomies and the pneumonectomies. As a rule, this leakage of air has its origin in alveoli or small bronchi and is attributed to "alveolopleural fistulae" by Johnson and Kirby,³ but it may also be due to imperfect bronchial stump closure. The postoperative management is designed to re-expand the lung to bring about apposition of tissues and thereby to promote healing. With good response, suction can be discontinued in forty-eight to seventy-two hours, but if re-expansion of the lung is slow, suction for as long as three weeks is occasionally required.

Apical pneumothorax pockets may persist despite efforts at re-expansion. Observations by needle aspiration have demonstrated that some may have a small bronchial opening.

Pneumothorax may recur two to ten days after suction is discontinued and on one occasion in our experience it developed after seven weeks. These pneumothoraxes are usually due to "late leaks" or "alveolopleural fistulae," although they may be a manifestation of a true bronchopleural fistula. The distinction between these types of air leakage has not been clearly defined, but appears to be real. It is suggested that in future studies they should be reviewed separately. In our experience, the "late leaks" may be asymptomatic with pneumothorax detected only on routine fluoroscopy, they may be noted by the patient as "noises in the chest," or they may cause significant dyspnea. On the other hand true bronchopleural fistula is related to imperfect healing of a bronchial stump and is marked by continued free flow of air, failure of lung to expand, and the frequent development of empyema as a complication.

Apical pneumothorax pockets occurred in 12 patients. 9 received no treatment and have had no complications, 2 were treated by thoracoplasty, and 1 was treated by thoracotomy, decortication, and repair of bronchial opening.

Late leaks occurred in 30 patients and responded satisfactorily to either needle aspiration or catheter suction. These, in conjunction with the 12 apical pockets accounted for 42 postoperative pneumothorax problems. Forty occurred after elective operations and accounted for one half the complications occurring in this group. Pneumothorax complicated lobectomy in 2 instances, segmental resection in 28, and subsegmental resection in 12.

Bronchopleural fistula occurred in 7 patients and was complicated by tuberculous empyema in 4 and nontuberculous empyema in 3. The management and course is discussed in the paragraphs on empyemata.

The incidence of alveolopleural fistulas has decreased with a change in management of the raw lung surfaces. The initial desire to avoid distortion of lung required leaving the surfaces unapposed. The occurrence of late leaks led to a policy of approximating the pleural edges by interrupted silk sutures at the site of local excisions or subsegmental resections. The next step was the apposition of the raw surfaces left by segmental resections whenever possible. A limited distortion of the lung was accepted as the price of a less complicated convalescence.

(b) The problem of fluid in the pleural space is more serious than postoperative pneumothorax

Nontuberculous empyema occurred in 8 patients. It was limited in extent in 4, 2 of whom responded to antibiotics and 2 to simple drainage. An infected hemothorax following pneumonectomy responded to varidase and antibiotics. Bronchopleural fistula was a complicating and probably causative factor of empyema in the remaining 3, treatment took the form of thoracotomy closure of fistula, and limited thoracoplasty and was successful in all, although 1 patient required a second operation.

Tuberculous empyema occurred in 4 instances—always in association with a bronchopleural fistula. Lobectomy was successful in 1 and failed in 1, a third patient recovered following drainage, and the fourth following thoracoplasty.

Hemothorax occurred after 12 operations. Persistent postoperative bleeding required open thoracotomy for control of bleeding on 2 occasions, 1 patient recovered uneventfully, the second died of overtransfusion. Of the remainder decortication was used once and varidase on 3 occasions, while 6 patients recovered without intervention but with the expected local pleural thickening. It is our impression that this complication can be minimized by care in dealing with adhesions. Bleeding from pulmonary vessels is, in our experience, a less common cause. Hemothorax of significant degree merits prompt attention.

Uninfected fluid accumulations of significant extent occurred in 5 patients and all responded well to aspiration.

Bronchial Tree—The airways were a less frequent site of complications. Retained secretions required bronchoscopy in 11 patients. This relatively low incidence reflects good postoperative nursing care and the successful use of endotracheal catheter suction. Only 1 instance of spread was noted in the area of retained secretions.

Ulceration of the bronchial stump was observed in 2 patients. One had an exacerbation of disease while a second had progressive active disease and died.

Postoperative spread of tuberculosis was noted in 7 patients, 3 were in the homolateral lung and 4 in the contralateral. Three have recovered, 2 are currently active and 2 died of progressive disease.

Other Complications—Other complications noted include tuberculosis of the chest wall in 1. Nontuberculous bronchopneumonia occurred 3 times, all patients recovering. Nontuberculous chest wall infections of limited extent occurred in 3. Unintentional interruption of the phrenic nerve occurred in 2 and thrombophlebitis in 1.

Significant complications occurring later than the sixty day postoperative period are tuberculosis of the chest wall twice at seven and eighteen months and 5 exacerbations of pulmonary tuberculosis observed between four and nine months.

THE AFTER-HISTORY

The first section of this paper has dealt with the "risks" of resection surgery. It is now proposed to assess the benefits of the procedure. This type of surgery has been carried out in the belief that necrotic lesions constitute the source of spreads and relapses and that the excision of such lesions results in freedom from exacerbation of tuberculous disease. The degree of such freedom should be complete if all lesions are removed and relatively good if all filled or open cavities and major areas of disease are removed. Medlar⁴ has shown that tuberculous lesions in the lower portions of pulmonary lobes seldom progress to liquefaction and cavity formation, unless they are large. Hence, residual lesions located in the dependent portions of the lobes should have a better prognosis than the lesions of similar size that are located in the apical and posterior segments of the upper lobes and the superior segments of the lower lobes.

In our early experience, streptomycin was given alone with the hope that it would provide bacteriostasis during the operative period. It soon became evident that long courses of streptomycin, para-aminosalicylic acid, or isoniazid in any combination aided the process of resolution of non-necrotic tuberculosis and thereby decreased the volume of diseased lung requiring resection. Accordingly, x-ray stability became a guide to the optimum time for operation, but as experience accumulated, shorter periods of preoperative stability came to be accepted as adequate. With the exception of the early short-term therapy, drug treatment in preparation for surgery was attended by relatively uniform beneficial effect as evidenced both by the pathologic studies and the postoperative course.

It was later recognized that, in spite of long-term drug therapy, areas of new disease are demonstrable in resected specimens and drugs, therefore, do not completely prevent extension of tuberculosis despite negative culture findings on sputum or gastric washings. As a corollary, spread during operation may occur. This observation was regarded as an indication for the use of drugs for an adequate postoperative period, the aim being to secure resolution of any new area of disease before necrosis develops. The more recent cases have, therefore, had a minimum of three to four months of postoperative drug administration.

The follow-up data will be presented in accord with the anniversary method of Bosworth and Alling,⁷ which affords a continuing picture of the after-history. The present review is of necessity a preliminary report, as five to ten years of follow-up will be required to give the full story. Studies of both thoracoplasty cases (Douglass⁵) and minimal cases (Lincoln and associates⁶) have shown that relapses continue to occur during and after the ten-year period. The hazard of relapse is greatest, however, during the first three years after reaching an arrested status and this follow-up covering the first two postoperative years should give an indication of the value of these resection operations.

TABLE VI PATIENTS RECEIVING RESECTION SURGERY TO SHOW AFTER HISTORY SURGERY, STATE OF DISEASE, AGE, AND SEX

TABLE VI PATIENTS REQUIRING RESECTION		TO SHOW AFTER RESECTION						2, A. EFFECTIVE WITHIN RESIDUAL DISEASE						2, B. EFFECTIVE WITHOUT RESIDUAL DISEASE											
		TOTAL RESECTION PATIENTS						I. REMAINING PATIENTS						TOTAL						TOTAL					
		TOTAL		M		F		TOTAL		M		F		TOTAL		M		F		TOTAL		M		F	
ALL AGES	1	507	100	291	113	113	110	2	18	60	256	10	165	51	111	58	81	2	111	58	81	2	1	2	
M	1	210	10	145	51	51	53	1	25	27	118	14	91	23	95	25	10	1	95	25	10	1	1	1	
F	1	271	90	149	62	62	57	1	23	33	138	20	81	28	70	33	12	1	70	33	12	1	1	1	
0-19	1	26	11	13	2	2	-	-	-	-	13	3	8	2	11	8	5	-	11	8	5	-	-	-	
20-29	1	31	13	17	1	1	5	-	3	2	16	4	10	2	13	9	1	-	13	9	1	-	1	1	
30-39	1	42	12	50	20	20	16	-	7	9	31	5	20	10	22	7	11	-	22	7	11	-	-	-	
40-49	1	112	27	65	20	20	21	1	10	10	57	12	35	10	31	11	20	-	31	11	20	-	-	-	
50 and over	1	81	8	42	11	11	15	-	7	8	28	2	23	3	18	6	12	-	18	6	12	-	1	1	
	1	91	17	45	22	22	19	-	7	11	42	7	25	10	21	10	13	-	21	10	13	-	-	-	
	1	12	1	27	11	11	14	-	8	6	19	2	12	5	9	2	7	-	9	2	7	-	-	-	
	1	33	2	19	12	12	9	-	2	7	19	2	12	5	5	-	5	-	5	-	5	-	1	1	
	1	25	5	13	7	7	9	1	3	4	14	2	9	3	7	2	1	-	7	2	1	-	-	-	
	1	9	1	3	1	1	1	-	1	3	1	1	2	1	-	-	-	-	-	-	-	-	-	-	

The after-history of the (1) salvage group will obviously differ from the elective group and will be presented separately. The elective group will be subdivided into (2,a) those in whom only the major foci were removed, and (2,b) those in whom all recognizable disease was removed. The composition of each group by age, sex, and stage of disease is shown in Table VI.

In each subgroup the patient's status will be reported as (1) "Satisfactory" if sputum and gastric washing examinations (excluding the first seven postoperative days) were consistently negative and chest roentgenograms remained stable (Essentially equivalent to Inactive, N T A Standards, 1950).

(2) "Doubtful" if one to three isolated reports of a low-grade positive concentration or culture are recorded, the x-ray films being stable (Essentially equivalent to Arrested, N T A Standards, 1950). This category is designated because of uncertainty as to the significance or validity of the findings. The limited laboratory data did not warrant the conclusion that active disease was present. The after history of these individuals is revealing. Thus far, only 1 has shown evidence of active disease at the next anniversary, the remainder being well.

(3) "Active" if positive sputum persists or recurs, or x-ray film shows evidence of active disease.

(4) "Deaths" occurring within sixty days are recorded as postoperative, others as late.

Salvage Cases—Table VII shows the indications for operation and the follow-up results at six months. Of the 55 patients with thoracoplasty failure, 32 were classified "satisfactory" and 8 "doubtful" six months following operation. This suggests that among selected cases of thoracoplasty failure, three-quarters may be expected to be salvaged by resection surgery. The follow-up data of the cases of persisting cavity indicate that an equivalent salvage may be anticipated. Patients with empyema or bronchostenosis generally had a favorable course. The highest mortality occurred among the cases of destroyed lung although the 50 per cent salvage is gratifying.

TABLE VII SALVAGE GROUP INDICATION FOR OPERATION, STATUS AT SIX MONTHS

INDICATION	TOTAL	SATISFACTORY	DOUBTFUL	ACTIVE	DEAD
Total	110	67	14	18	11
Thoracoplasty failure	55	32	8	12	3
Persisting cavity, 1 cm	31	17	6	4	4
Empyema	7	6	—	—	1
Bronchostenosis	9	8	—	1	—
Destroyed lung	8	4	—	1	3

Fifteen deaths occurred in the group of 110 patients. 12 of them within sixty days of operation. The results in relation to type of operation and period of follow-up are shown in Table VIII.

The over all outcome for this group is shown in Fig. 2. This depicts the status of all patients at six months and the expected status at one and two years based on the assumption that the patients with shorter follow-up will

TABLE VIII SALVAGE GROUP TYPE OF OPERATION AND AFTER HISTORY STATUS

PROCEDURE	NUMBER OF PATIENTS	OPERATIONS CONTRALATERAL	SECOND OPERATION ON SAME SIDE	TOTAL OPERATIONS
Pneumonectomy	36	—	—	36
Lobectomy	54	—	3	57
Segmental	14	1	3	18
Subsegmental	6	3	—	9
Total	110	4	6	120

STATUS AT SIX MONTHS AFTER COMPLETION OF SURGICAL PROGRAM

	LUNG	LOBE	SEGMENT	SUBSEGMENT	TOTAL
Satisfactory	24	32	7	4	67
Doubtful	3	9	2	—	14
Active	3	10	5	2	20
Dead	6	3	—	—	9
Total	36	54	14	6	110

STATUS AT TWELVE MONTHS AFTER COMPLETION OF SURGICAL PROGRAM

	LUNG	LOBE	SEGMENT	SUBSEGMENT	TOTAL
Satisfactory	19	29	3	1	52
Doubtful	3	1	—	—	4
Active	2	10	3	2	17
Dead	—	—	2	—	2
Incomplete*	6	11	6	3	26
Total	30	51	14	6	101

STATUS AT TWENTY FOUR MONTHS AFTER COMPLETION OF SURGICAL PROGRAM

	LUNG	LOBE	SEGMENT	SUBSEGMENT	TOTAL
Satisfactory	20	21	2	1	44
Doubtful	—	1	—	—	1
Active	2	7	2	1	12
Dead	—	1	—	—	1
Incomplete*	2	10	2	1	15
Total	24	40	6	3	73

Incomplete These patients operated on too recently to allow observation at this anniversary

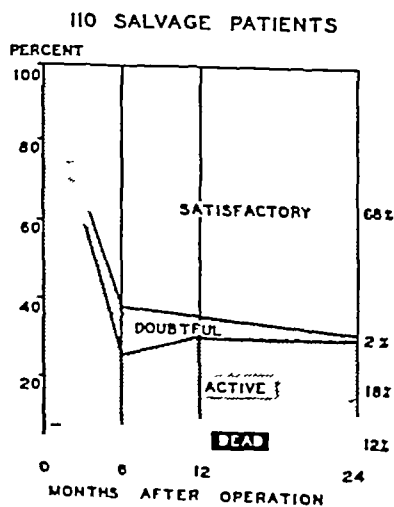


Fig. 1. After history of 110 salvage patients showing status at six, twelve, and twenty four months after operation

TABLE IX. SAVVAGE GROUP (CALCULATIONS MADE FROM TABLE VIII TO PLOT ANNIVERSARY STATUS FOR FIG. 2)

ANNIVERSARY	LIVING AT ANNIVERSARY			DIED SINCE PREVIOUS ANNIVERSARY	TOTAL	INCOME FIFTY	FACTORS		EXPECTED PERCENTAGES			
	SATISFACTORY	DOUBTFUL	ACTIVE				PROVISIONAL	CORRECTION	SATISFACTORY	DOUBTFUL	ACTIVE	ACCUMULATED DEAD
0					110			0.0001				
6 mo	67	14	20	9	110	-	1.0000	0.9091	60.9	12.7	18.2	8.2
12 mo	72	1	17	2	75	26	1.3467	1.2243	63.7	4.9	20.8	10.6
24 mo	41	1	12	1	58	15	1.2586	1.5409	67.9	1.5	18.4	12.1

have a course comparable to those actually observed. The calculations made from Table VIII in order to plot the status at anniversary are shown in Table IX.

Elective With Residual Disease—The follow-up of the group of 256 elective cases with known or questionable residual disease is presented in Table X which shows the after-history in relation to the type of operation and duration of follow-up. It is noted that 4 deaths have occurred, and that the number with doubtful status declines with time. Six of the active cases had persisted in this status from operation while 2 represent relapses, both homo-lateral.

The over all picture is shown in Fig. 3.

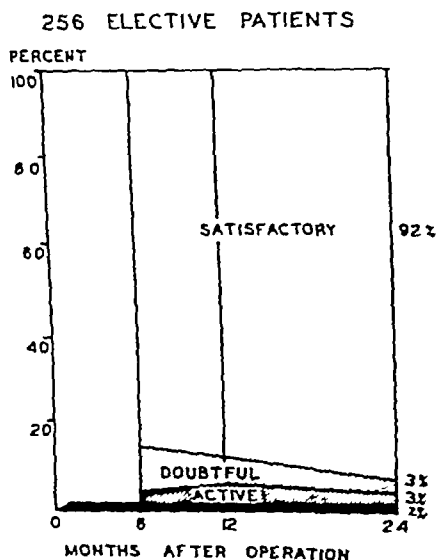


Fig. 3—After history of 256 elective patients with residual disease showing status at six, twelve and twenty four months after operation.

Elective No Residual Disease—This group of 141 patients is designated for separate analysis because of uniformity of observation both in palpation of lung tissue, type of x-ray examinations and study of resected specimens. It is believed that all palpable disease in the lungs operated upon was removed and that the opposite lung showed no x-ray lesion (plaingrams having been made). If the thesis is valid that necrotic lesions are the lurking place of tubercle bacilli and hence are the cause of exacerbations, then this special group should be free from future manifestations of tuberculosis or at worst should have a low incidence of relapse.

The postoperative clinical course of the 141 patients has been quite favorable. At six months 1 patient had had four positive concentrates and was placed on drug therapy with subsequent conversion of sputum. This was the only patient regarded as a failure. Five others were at some time in 'doubtful' status. 1 had one bacillus on concentration on one occasion, 2 had one

colony on culture on one occasion, a fourth had three cultures of acid-fast organisms one of which on test failed to kill a guinea pig, while the fifth, at the second year had one colony on culture which failed to infect a guinea pig. The procedures used and follow-up in this group are recorded in Table XI, 40 patients having had bilateral operations. No figure is needed.

TABLE X. ELECTIVE GROUP 2, A. PATIENTS WITH RESIDUAL DISEASE, TYPE OF OPERATION, AND AFTER HISTORY STATUS

PROCEDURE	NUMBER OF PATIENTS	OPERATIONS CONTRALATERAL	SECOND OPERATION ON SAME SIDE	TOTAL OPERATIONS	
Pneumectomy	3	—	—	3	
Lobectomy	50	3	—	53	
Segmental	93	11	1	105	
Subsegmental	110	16	1	127	
Total	256	30	2	288	
STATUS AT SIX MONTHS AFTER COMPLETION OF SURGICAL PROGRAM					
	LUNG	LOBE	SEGMENT	SUBSEGMENT	TOTAL
Satisfactory	3	42	79	93	217
Doubtful	—	7	9	11	27
Active	—	—	3	4	7
Dead	—	1	2	2	5
Total	3	50	93	110	256
STATUS AT TWELVE MONTHS AFTER COMPLETION OF SURGICAL PROGRAM					
Satisfactory	3	19	45	77	144
Doubtful	—	4	2	5	11
Active	—	—	3	4	7
Dead	—	—	—	—	—
Incomplete*	—	26	41	22	89
Total	3	49	91	108	251
STATUS AT TWENTY FOUR MONTHS AFTER COMPLETION OF SURGICAL PROGRAM					
Satisfactory	1	16	22	21	60
Doubtful	—	—	1	1	2
Active	—	—	—	2	2
Dead	—	—	—	—	—
Incomplete*	2	7	27	62	98
Total	3	23	50	86	162

*Incomplete. These patients operated on too recently to allow observation at this anniversary.

Fifty-eight of these had minimal lesions, 81 were moderately advanced, while only 2 were far advanced. Twenty-one had never had tubercle bacilli demonstrated. The surgical judgment displayed in operating on these may be open to discussion.

A review of the pathologic reports however shows that among the 21 patients who had never had positive sputum one cavity remnant was demonstrable while 11 had necrotic lesions 1 cm. or more in size. All but 1 had definite necrotic lesions—this patient showing scarring and focal pneumonia. Of the remaining 120 patients 65 fifteen of them minimal had evidence of open or filled-in cavity, bronchial communication being demonstrated by wire probe or random sections in 27. Four others had bronchial debris indicating a connection. This comprises with 98 bronchial communications demonstrated in 141 filled-in cavities found in advanced cases and studied intensively by Medlar.¹ One other point of interest in the review is the fact that abnormal

bronchi were noted in 10. This compares with the finding of concomitant tuberculosis and bronchiectasis in 26 of a series of 295 specimens studied by Medlar.⁵

It is also significant that in the group of 58 minimal cases 1 had the major focus in the middle lobe, while in all others the major focus occurred in the apical and posterior segments of upper lobes or the superior segments of the lower lobes. When all excisions including calcified nodules were counted three fourths were in the superior portions. This is consistent with Medlar's⁵ findings in necropsy studies.

TABLE XI ELECTIVE GROUP 2, B. PATIENTS WITH NO RECOGNIZED RESIDUAL DISEASE, TYPE OF OPERATION AND AFTER HISTORY STATUS

OF OPERATION AND AFTER HISTOPATHIC STATUS					
PROCEDURE	NUMBER OF PATIENTS	OPERATIONS CONTRALATERAL	SECOND OPERATION ON SAME SIDE	TOTAL OPERATIONS	
Pneumectomy	1	-	-	1	
Lobectomy	15	-	-	15	
Segmental	74	18	-	92	
Subsegmental	51	22	-	73	
Total	141	40	-	181	
STATUS AT SIX MONTHS AFTER COMPLETION OF SURGICAL PROGRAM					
	LUNG	LOBE	SEGMENT	SUBSEGMENT	TOTAL
Satisfactory	1	14	71	50	136
Doubtful	-	1	2	1	4
Active	-	-	1	-	1
Dead	-	-	-	-	-
Total	1	15	74	51	141
STATUS AT TWELVE MONTHS AFTER COMPLETION OF SURGICAL PROGRAM					
Satisfactory	1	14	45	29	89
Doubtful	-	-	-	1	1
Active	-	-	-	-	-
Dead	-	-	-	-	-
Incomplete*	-	1	20	21	51
Total	1	15	74	51	141
STATUS AT TWENTY FOUR MONTHS AFTER COMPLETION OF SURGICAL PROGRAM					
Satisfactory	1	4	14	8	27
Doubtful	-	-	1	-	1
Active	-	-	-	-	-
Dead	-	-	-	-	-
Incomplete*	-	10	30	22	62
Total	1	14	45	30	90

*Incomplete. These patients operated on too recently to allow observation at this anniversary.

BACTERIOLOGY

No data on the bacteriology of excised tissue are presented because this problem is in a state of flux. Bacteriologists recognize the need for research studies. It can be stated however that whenever thorough search of necrotic lesions for tubercle bacilli was carried out they were always demonstrable in smears. Medlar⁵ studied 376 necrotic lesions in resected specimens from 145 patients. If only one necrotic lesion from a patient was examined two thirds were positive; when three or more lesions were examined tubercle bacilli were found in the excised tissue from every patient.

SUMMARY

1. Five hundred seven patients had 589 resection procedures for tuberculosis.

2 A salvage group of 110 patients had 120 operations with 11 postoperative deaths and 3 late deaths. There were 13 serious and 9 minor postoperative complications. Eleven tuberculous complications occurred.

3 An elective group of 397 patients had 469 operations with 4 postoperative deaths. There were 16 serious and 67 minor postoperative complications. One tuberculous complication occurred.

4 Study of after-history gave the following results:

- (1) The salvage group of 110 patients shows that at two years 70 per cent were well, 18 per cent were active, and 12 per cent were dead.
- (2,a) The elective group of 256 patients with residual disease shows at two years 95 per cent well, 3 per cent active, and 2 per cent dead.
- (2,b) The elective group of 141 patients with no recognized residual disease shows at two years no deaths and no patient with active disease.

5 A review of the pathology of the 141 patients in the latter group showed necrotic lesions present in all but 1, while 65 had evidence of open or filled-in cavity.

We wish to express appreciation for the interest and help given by Dr Ramon Larios Contreras, Dr Joseph J Krejci, Dr Dennis M Rosenberg, and Dr Ralph V Ramirez in analyzing the case histories, and to Dr Ramirez for his review of the pathology.

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THE COORDINATION OF SURGERY AND COMBINED CHEMOTHERAPY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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PROLONGED combined chemotherapy with various antituberculosis drugs in varying regimens is still by no means adequately evaluated. Since late 1946 when the first patients receiving prolonged streptomycin and PAS therapy were studied,¹ an attempt has been made to use only clear-cut, gross, and simple criteria for evaluation of this and other therapeutic regimens. Variables introduced by adjunctive collapse measures have purposefully been kept to a minimum. The great individual variable of bed rest has been practically excluded by the restless nature of the young veteran as a patient.

Thus, by using the criteria of positive or negative sputum cultures, open or closed lesions by rigid radiologic examination, and the occurrence or absence of relapse, an attempt has been made to evaluate the effectiveness of chemotherapy *alone*.

Stimulated by Dr. Medlar's evidence²⁻³ that closed, caseous, necrotic foci persisting after all types of therapy are very probably the "seed beds of relapse," an active program of resection of these and other residual lesions has been carried on concurrently. Through reasons of selection, which at first were largely accidental and only recently have become in part purposeful, there have been collected two series of patients. Both were treated to presumed maximal benefit with chemotherapy. Then one group was resected, and one was not. The clinical aim generally was to carry the patient to maximal benefit on chemotherapy and then, if possible, to resect any residual lesions with a view to preventing relapse.

MATERIAL

Fig. 1 demonstrates the effectiveness of original streptomycin and PAS therapy on 179 consecutive cases of *sputum-positive, cavity tuberculosis*. Using only the criteria of sputum bacteriology on culture and the presence or absence of open lesions by roentgenography including periodic planography we develop three categories. The Open-Positive group, which constituted 100 per cent at the onset of therapy, is 23 per cent after eight months of continuous combined drug treatment. However, although 77 per cent of the total had converted their sputa for three months or longer during eight months of therapy, only 55 per cent had closed all their open lesions. A portion of the continued closure and conversion rate increase noted after eight months is an artifact due to removal from the study by surgical intervention, of a few cases felt most unlikely to achieve cavity closure.

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The material presented herein represents the opinions of the authors and does not in any way reflect the policies of the Medical Director of the Veterans Administration.

The phenomenon of sputum conversion for three months or longer, as associated with failure to close all lesions, yields the group of Open Negative patients. These constitute from 20 per cent to 40 per cent of the total at various periods throughout twelve months of chemotherapy.

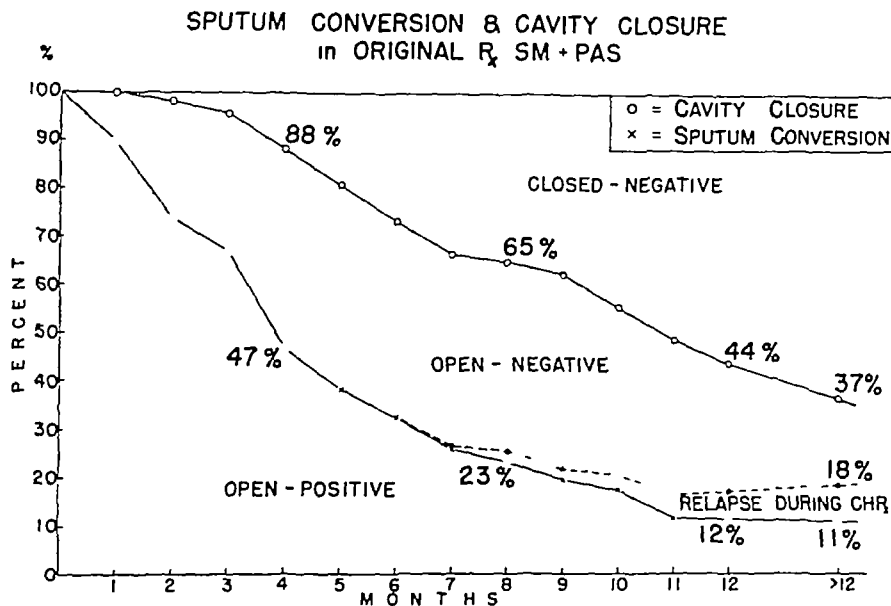


FIG 1

The patients who close all their lesions always convert their sputa as well. Thus Closed Negative group ultimately comprises about 50 per cent of the cavitary lesions treated. The phenomenon of sputum relapse during combined chemotherapy noted in the Open-Negative group is not observed in the Closed-Negative group. It is worth noting that a concurrent series of 89 consecutively treated patients who *did not* have cavity at the onset of chemotherapy all converted their sputa in less than eight months, and none of these relapsed during continued drug treatment. This type of case swells the Closed-Negative group in any clinical series and, in so far as we can determine, Closed-Negative patients behave exactly alike by our criteria, whether they closed during therapy or were not open at the onset of therapy.

Failure to convert sputum or relapse to positive during continued chemotherapy is *always* associated with persistence of open lesions.

The next item we feel important to determine, after establishing these three categories of response noted during prolonged combined therapy, is the fate of each after discontinuation of the drugs.

The arbitrary minimal period of eight months of therapy chosen for the unselected cases in Table I was exceeded by many months in most. We know of no convincing data delineating the optimum duration of effective combined chemotherapy.

We perhaps have had exceptionally poor luck with our patients who failed to convert the Open Positive group. This may be because they refused, or

TABLE I. FOLLOW UP—UNRESECTED (ORIGINAL STREPTOMYCIN AND PAS EIGHT MONTHS OR LONGER)

STATUS AT DISCONTINUATION OF CHEMOTHERAPY	NO PATIENTS	DEAD OF TUBERCULOSIS	NOW ACTIVE	NOW INACTIVE OR NONACTIVE	% INACTIVE OR NONACTIVE
Open Positive	25	7	18	0	0
Closed Negative	57	0	3	54	95
Open Negative	27	4	14	9	33

were unsuitable candidates for, any adjunctive measures. Most of those with significant follow-up antedated the availability of the newer antituberculosis drugs. Of 25 patients who had original treatment with streptomycin and PAS for eight months or more, and who failed to convert and close, 7 are dead of tuberculosis and 18 have continuing active disease.

Of 57 who achieved a Closed-Negative status with the same chemotherapy program, 95 per cent are inactive or nonactive for periods up to four years after discontinuation of their drugs.

Of the 27 patients given original streptomycin and PAS therapy for eight months or longer, who converted their sputum but demonstrated residual open lesions radiologically at the time the drugs were discontinued, 18 relapsed to a frankly active state, mostly within one year. This phenomenon of relapse after chemotherapy was suggested in Fig. 1 by the small, but definite, relapse incidence noted during continued chemotherapy among these Open-Negative patients.

This is a necessary type of base line data, admittedly still scanty in numbers upon which we must evaluate the therapeutic effectiveness of surgical intervention both now on a short-term basis and as years of follow-up accumulate. If we do not place our cases in some such categories, we cannot ever hope to untangle the mass of clinical reports already accumulating. When one tries to evaluate reports of several hundred cases apparently of all three categories resected during chemotherapy, it is confusing to know that relapse of patients treated to a Closed-Negative state and *not resected* is very rare.

Table II presents follow-up data on the three groups resected. The same data shown in Table I are again included with each category for convenient comparison.

TABLE II. FOLLOW UP—RESECTED VS. UNRESECTED

STATUS AT RESECTION OF PULMONARY CHEMOTHERAPY	NO PATIENTS	DEAD OF TUBERCULOSIS	NOW ACTIVE	NOW INACTIVE OR NONACTIVE	% INACTIVE OR NONACTIVE
Open Positive	45 (7 bilateral)	2	8	35	78
Unresected*	25	7	18	0	0
Closed Negative	214 (64 bilateral)	0	4	210	98
Unresected*	57	0	3	54	95
Open Negative	96 (19 bilateral)	0	3	93	97
Unresected*	27	4	14	9	33

* Patients on chemotherapy 6 months or longer before drug discontinuation.

The salvage of 78 per cent thus far in the Open-Positive resected group is very encouraging. However, 22 per cent of some type of clinical failure still leaves a great deal to be desired. The resected and unresected Closed-Negative cases as yet show no significant differences in follow-up. The rationale for resecting these closed residua originally put forth before this Association by Drs. Ryan, Medlar, and Welles in 1951² is still very appealing in many ways. If we are in any doubt about the effectiveness of new drug combinations, or the degree of drug sensitivity at the onset of therapy, we still feel it is safer by far to resect residua of this type until prolonged observation of unresected controls demonstrates they may be safely left in. Lest time find us with inadequate data on drug-sensitive patients who achieve the Closed-Negative state, we still urge that at least a portion of such cases be resected in those institutions qualified for adequate clinical follow-up.

It is with the Open-Negative patients that we have found the most gratifying results of resection during prolonged chemotherapy. The marked degree of bacterial suppression in these cases, despite the latent tendency to relapse noted in the unresected patients, allows resection with few or no tuberculous operative complications. The salvage of 97 per cent as compared to 33 per cent of the unresected group gives us currently a rather clear cut operative indication.

TABLE III. COMPLICATIONS IN 463 THORACOTOMIES IN 373 PATIENTS INCLUDING 10 PNEUMONECTOMIES, 95 LOBECTOMIES, 147 SEGMENTALS, 211 WEDGES

NONTUBERCULOUS		TUBERCULOUS	
Operative deaths	3* (0.6%)	Spread	2*
Torn pulmonary artery requiring pneumonectomy	2*	BPF with tuberculous empyema	2*
Space requiring thoracoplasty	14*	Stump ulcer	2*
Subscapular infection	3*	Tuberculous infection pulmonary operative defect	2*
Bleeding requiring SK-SD or reoperation	18		
Transient BPF closed drainage only	50		
BPF treated by thoracotomy	3		
Psychosis	3		
Phrenic section	2		
Homologous serum jaundice	1		
Transient hemiplegia	1		

*These complications yielding a lasting deficit totaled 40 or 6.5% (includes operative deaths). Open Positive 1.2%, Open Negative 6%, and Closed Negative 5%.

SK-SD = streptokinase-streptodornase

BPF = bronchopleural fistula

COMPLICATIONS

It is necessary that operative mortality and morbidity be considered in any evaluation of surgical interference. We have not found these factors in any way prohibitive. Table III is a simplified listing of operative mortality and significant operative morbidity encountered in 463 thoracotomies involving 373 patients. These included as the major operative procedure 10 pneumonectomies, 95 lobectomies, 147 segmental resections, and 211 wedges. Of the three deaths, one patient expired of the complications of a lower nephron nephrosis after lobectomy, one died immediately following biopsy of a lung involved with both tuberculosis and an Auerz-like complex, and one died of

diffuse uncontrollable hemorrhage during a wedge resection. We want to direct your attention to those complications, marked with the asterisk, which were felt to leave the patient with some lasting defect not anticipated when resection was undertaken. These include the operative deaths, the extension of the resection to pneumonectomy because of an irreparable operative accident, thoracoplasty for postoperative space problems, subscapular infection, and all the tuberculous complications as listed, spread, fistula with specific empyema, tuberculous stump ulcers, and two tuberculous infections of very localized operative defects within the parenchyma. The fact that six of the total of eight tuberculous complications occurred in the Open-Positive group accounts for the only difference in significant complications in the three categories discussed.

DISCUSSION

Explanations for the high relapse rate in Open-Negative patients, unresected, are possibly complex. The only significant data available to us, however, come from the bacteriologic examination of resected material by culture and guinea pig inoculation. Table IV summarizes our findings in all three groups by these methods. It is not surprising that 98 per cent of the Open-Positive patients yielded a positive specimen. Of 204 patients resected after being closed and negative for at least three months, only 18 per cent have a positive culture or guinea pig from the specimen despite the fact that over 80 per cent were positive on smear.

TABLE IV BACTERIOLOGIC DATA ON RESECTED LESIONS

	NO OF PATIENTS	NO OF PATIENTS WITH POSITIVE SPECIMEN BY CULTURE OR GUINEA PIG	% POSITIVE
Open Positive	44	43	98
Open Negative	92	37	40
Closed Negative	204	40	18

The Open Negative group, similarly studied, yielded growth of tubercle bacilli in 40 per cent of patients. The reasons for negative sputa in these patients may be simply mechanical. Why only 40 per cent of this category produced a positive culture while 66 per cent of similar patients unresected relapsed may be explained in part by the fact that we now feel obliged to resect and include in the Open-Negative group open lesions which may occasionally prove to be blebs, pneumatocoles or cystic bronchiectasis. We do not feel we can safely differentiate these lesions from frank residual cavities by current clinical and radiologic techniques. Bronchography has as yet proved of little assistance.

If we accept the apparent clinical application of these data, and choose to interfere surgically with those cases initially susceptible to chemotherapy but failing to close their cavities during treatment, the next question is that of timing. The following three factors seem important to us in the timing of resection during chemotherapy.

First, allowing time for maximal resolution of reversible disease will permit resection of a minimal amount of lung tissue. The incidence of significant

operative complications decreases in direct proportion to the magnitude of the resection. Furthermore, the substitution of a wedge or segmental resection for a lobectomy allows us to dodge the uncertain issue of overexpansion.

Resolution of tuberculous pneumonia is generally complete by four months of effective chemotherapy. The further slow clearing sometimes observed to continue for many months may be variously interpreted as resolution of the granulomatous or "proliferative" manifestations of the disease sloughing of necrotic foci through bronchial communications, or the inspissation of necrotic foci.

In any case, patients with significant pneumonic components probably should be allowed a minimum of four months of chemotherapy before resection is considered. Continued appreciable resolution from month to month may encourage further delay.

The second factor in timing is bacterial. Resections carried out during periods of bacterial suppression seem to incur almost no tuberculous operative complications. Clinically, our experience suggests that we utilized this factor in our Open-Negative and Closed-Negative cases. In the Open-Positive group both the period of maximal bacterial suppression and that of susceptibility of the patient's organisms to the drugs used had generally been passed when surgical interference was elected. The Open Positive group resected, also includes certain patients with open lesions whose sputa were negative for three or more months before relapsing to positive during combined chemotherapy. Therefore, it would appear that in the persistently Open patients whose quantitative sputum bacteriology declines rapidly to negative, resection if feasible should be carried out before the occasional "escape" of the organisms. This occurred no earlier than seven to eight months after onset of chemotherapy. (See Fig 1)

In the patients who never convert, there is almost always a period at about four months of chemotherapy when the bacterial population of the sputum is very low and still drug susceptible. This is succeeded after a period of usually no less than two months by an increasing population of increasingly resistant organisms.

Fig 2 shows the incidence of heavy positive sputum cultures during original streptomycin and PAS treatment. Note that at four months there are none with persisting heavy growth. The INH curve is superposed for comparison.

Fig 3 shows the rate of development of resistance to therapeutic concentrations of streptomycin and/or PAS during original treatment of 59 patients who failed to convert. This is a retrospective study of the Open Positive group. By six months, over 50 per cent were already showing significant resistance.

The third factor worthy of some consideration in the question of timing resection is that of cavity closure. If we are to draw any conclusions from our experience with resection of closed lesions residual after chemotherapy, we may say that perhaps it will ultimately be shown that they need not be removed. Therefore, it seems practical to explore vigorously the value of adjunctive collapse measures early in chemotherapy. These may include bed rest, pneumoperitoneum, and perhaps even a reevaluation of pneumothorax. Monaldi di un

age, with and without thoracoplasty, has proved of value in effecting closure of huge cavities during chemotherapy. If by these methods we can close a cavity before drug resistance has developed we can expect conversion to the Closed-Negative state. This would then direct evaluation of further surgical intervention to that category.

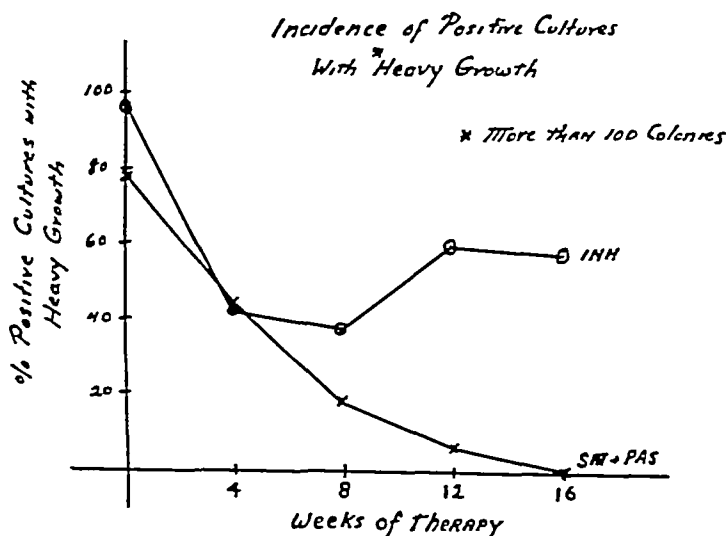


Fig 2

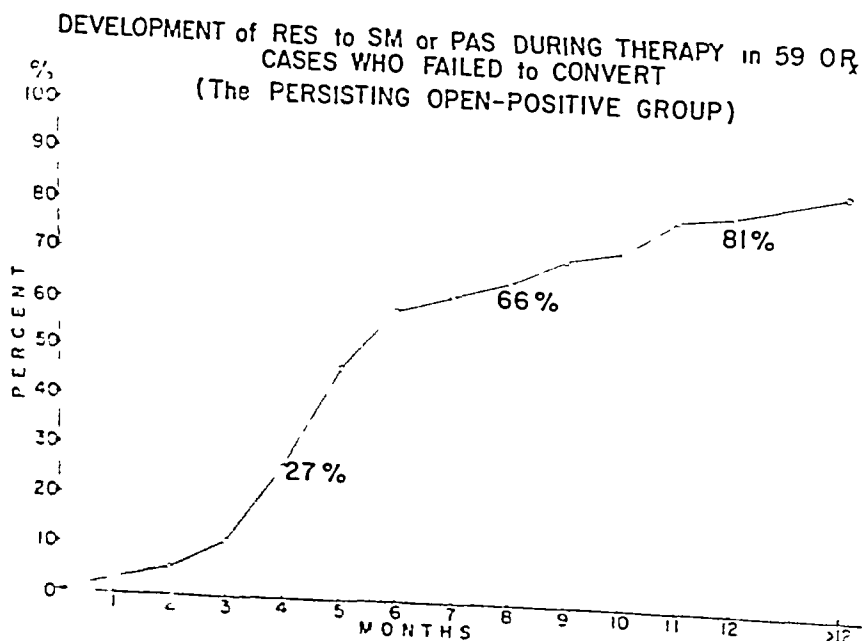


Fig 3

SUMMARY

In summary, the problem of resecting pulmonary tuberculosis during effective chemotherapy requires evaluation in the light of the response of the individual patient to chemotherapy. An attempt has been made to develop three clear cut clinically applicable categories of response for use in this evaluation. Factors which may influence the timing of various types of surgical interference have been mentioned.

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DISCUSSION

(Papers by Douglass, Bosworth, Judd and Chang, and Decker, Raleigh and Welles)

DR JOSEPH GORDON, Albuquerque, N M.—I should like to bring to your attention a point that has been helpful to us in the diagnosis of one of the complications of resection. This was the outcome of a study which was initiated at Rav Brook while I was there and has been transferred for continued study to the Veterans Hospital at Albuquerque, New Mexico. We started a program of trying to learn something about the pattern of pulmonary distension following resection to see what the bronchogram pattern would be and came across something that was rather helpful to us. The question is often raised as to whether or not the residual or recurring pneumothorax is associated with a bronchial fistula or is a residual pneumothorax which will slowly re expand. The usual criteria for making this diagnosis may be lacking or inconclusive. In films taken before and after resection we have found a pattern, which you will see on the four slides, that has been helpful and rather obvious.

(Slide) There is a residual pneumothorax in the upper left. In such cases it is often difficult by the usual means, which I shall not review here, to identify the presence of bronchial fistula.

(Slide) If a bronchogram is made you will get some clue as to whether or not there is a complicating bronchial fistula. The pattern of heavy oil which you see at the root is different from the pattern seen in the remaining expanded lung and there is a small blob of oil which has leaked out into the air space. The only possible means of this being present would be that there is a communication with this space and the bronchial tree.

(Slide) This is another case in which there is a residual pneumothorax pocket on the right side and here too we are concerned as to whether or not we are dealing with a residual pneumothorax space which will ultimately re expand or whether it is associated with a bronchial fistula. In each instance when this question arises, a bronchogram should be made.

(Slide) There is obvious evidence of a bronchial communication as noted in the pattern seen here and the oil free in the air pocket.

This gives us an opportunity to evaluate the problem and treat it immediately. In operating on these patients we have been able to identify the presence and location of the fistula, repair it and wind up with a satisfactory result.

DR JOSEPH W GAFF, Madison Wis.—We have been interested in this problem of pulmonary resection for the past decade and in 1948 we reported 80 consecutive resections with a conversion rate of 80 per cent. The patients were followed for a period of four years and the percentage of conversions held. A couple of years ago we reported 325 consecutive cases followed from one to seven years and our percentage of conversions, negative cultures, guinea pigs, etc, remained at 80 per cent. We are in the proc

no—I cannot as yet give you the statistics—of following 530 cases over a period of one to ten years. I thought it might be of interest to give some idea of what does happen over a decade in pulmonary resections.

(Slide) One very interesting thing. In the first 50 cases we reported 41 were thoracoplastic failures. In the 325 next reported 101 were thoracoplastic failures. Now it has dropped to less than 20 per cent as we have had more pointed therapy and we are now preserving more lung tissue. Only about one third of the first 50 patients had streptomycin because it cost nine dollars per gram and the hospital would not buy it. But this gives you some idea as to the mortality with the different types of operation. One out of 70 died following bilateral resection. The anesthetist left the pressure transfusion going and went away.

(Slide) Here are some of the complications bronchial fistulas etc. and this remained about the same all along. We had a total of 28 per cent fatal complications.

(Slide) A little more about bronchopleural fistula, it appears in about 5 per cent of cases in pneumonectomy, 4.5 per cent in lobectomy and 1 per cent in segmental resections. Some of these cleared spontaneously but most of them required further surgery and 7 of the 19 with bronchopleural fistulas died from sepsis, infection etc.

(Slide) Here are some of the complications which I will not enumerate but in the whole group they were 7.5 per cent.

DR WILLIAM M. LEES, Chicago, Ill.—I should like to report some of our experiences but before I do so I would like to make a plea that we utilize the same classification when we speak of patients with pulmonary tuberculosis. Dr Douglass mentioned a group of “salvage” and another group of “elective” patients and without knowing the exact figures I would state that we must have a different type of tuberculosis in Chicago because 55 to 60 per cent of our patients will be in his salvage group. I do not think that a patient with a 1 cm. cavity or with a thoracoplastic failure or with a destroyed lung who has a good lung on the other side is necessarily in a salvage group. With that introduction I would like to show a few slides illustrating the highlights of a study performed by Dr Adolfo Flores and Dr M. Lopez Beho who reviewed 250 consecutive resections performed at the Municipal Tuberculosis Sanitarium of Chicago, Illinois between Jan. 1, 1951 and Dec. 31, 1953.

(Slide) You will note that 70 per cent of the resections were in women, Negroes and whites are approximately even but more importantly, only 15 of the Negro patients had segmental or wedge resections. The vast majority of Negro patients had pneumonectomies or total lobectomies.

(Slide) Here you will note that the vast majority of patients had far advanced disease and it was bilateral in a great many cases, especially in those patients who had pneumonectomies. You will note that 60 of the 52 patients had bilateral disease. Many had bilateral cavities greater than 1 cm. some were 4 and 5 cm. in diameter.

(Slide) This shows our complications. The pneumonectomy group had an 85 per cent mortality, lobectomy 17 per cent and segmentectomies including wedges approximately 2 per cent. These data are rather closely allied to the figures Dr. Gale just presented.

In summary I would like to make a few comments which will in part concur with the statements made by the second author in that our complications may be lessened by giving combined chemotherapy for a minimum of four months, preferably six months, preoperatively. The lesions should be stable as demonstrated by x-ray film. The best results are obtained in patients who have negative sputum preoperatively. A careful preoperative evaluation of the patient's cardiopulmonary reserve and the bronchoscopic preparation of the bronchial tree are very important and careful bronchial closure with pleurodesis.

DR GARRETT P. SILEY, New York, N. Y.—The problem of residual pneumothorax after resection is a common one. Following upper lobe lobectomy, if the space is

not almost immediately filled by residual lung, I have used the following procedure in four cases. The patients have had a phrenic crush on the operative side within several days after operation, some in three days, and two between five and seven days. This has immediately closed the space or almost immediately, and it has remained closed. One patient was a female operated on almost two years ago. She remained well, went through a normal pregnancy, and is still well. This work was done on my service at Gouverneur Hospital in conjunction with Dr. Henry Dorfman, who made the original suggestion of postoperative immediate phrenic crush.

DR. RICHARD H. OVERHOLT, Brookline, Mass.—Since so much is known about the comparative hazards, that is, the risk of drug-treated disease and the risk of excisional therapy, I think it is time to concentrate, as the essayists have done, on the most appropriate time to carry out these various procedures. Dr. Wilson and Dr. Edward Hayes, Jr., have reviewed 231 consecutive primary drug cases treated in 1952 and 1953, and of this number, 108 were resected with less than thirty days of drug coverage. The remainder had preoperative coverage over a period of several months. Their studies revealed that there was no difference in the risk of operation or in the incidence of complications in the patients in whom the time of operation was set ahead to less than one month, as compared with those in whom the drug treatment was carried on preoperatively for longer periods. In fact, in this series of 231 consecutive cases of primary drug treatment, there was only one fistula and that was in a teen-age diabetic.

Just two comments about the problem of re-expansion of the remaining lung. We find it helpful to test the volume and shape of the expanded remaining lung with the ribs reapproximated in order to see if the reduced lung mass will fit its thoracic container. After an upper lobectomy or if combined with superior segmentectomy, the remaining group of segments, when expanded, assume the shape of a truncated cone. When expanded, the group of segments may wedge against the chest wall and not be able to find its way to the apex. Then, a modified type of thoracoplasty, performed simultaneously, will permit apical space obliteration.

One comment about tube management. (Slide) If two tubes are used and a Y connecting tube joins them to a single underwater seal, air from one part of the pleural cavity will find its way to another by way of the connecting tube. It is then difficult to bring the lung out. If both tubes are brought under the water seal separately, the obliteration of one part of the cavity is not influenced by the other. Since this simple change has been made, we have very little difficulty in obtaining immediate re-expansion and holding the lung out, even after the most complicated segmental resection.

DR. LOUIS R. DAVIDSON, New York, N. Y.—My remarks will be directed to the paper presented by Dr. Douglass; more particularly I shall confine my observations to that part of his paper which is called "after history." The thoughts which have just been presented to us have their being in bacteriology. However, in the evaluation of a patient relative to his final cure, which is surely a part of "after history," one must think not only in terms of bacteriology but also in terms of physiology, pathology, sociology, and economics. It is with these features in mind that I should like to refer to a recent paper which appeared in *Diseases of the Chest* in which our group at Sea View Hospital after analyzing 487 consecutive cases which had received excisional surgery evaluated them particularly as regards "after history." Our greatest patient load is about 80 per cent Negro and Puerto Rican. The major portion receiving excisional surgery had pneumonectomy performed. Of these, 53 women who have had excisional surgery have given birth to 67 children. Only one patient died and she was originally a calculated risk. One patient had pneumonectomy performed in the presence of contralateral pneumothorax and in addition gave birth to a child subsequent to surgery when still carrying a contralateral pneumothorax. At the moment she is gainfully employed.

This brief résumé is submitted in order to emphasize not only the pathologic status of the patient but also to draw attention to the vital necessity of evaluating the resulting physiologic and pathology together with the patient's sociologic and economic level.

DR JOSEPH A. WEINBERG, Long Beach, Calif.—I wonder if we all have the same understanding of what is meant by wedge resection and segmental resection. In addition to the anatomic consideration, segmental means to me that the pulmonary parenchyma is left exposed, and wedge means that the raw surfaces are pleuralized. This differentiation is important especially in dealing with tuberculous lesions. Wedge resection is a more surgical procedure than segmental resection because infection is less likely to occur with pleuralization of the naturally contaminated parenchyma, also, air leakage is less likely to occur if the exposed parenchyma is covered.

Dr Reiter of our clinic has provided me with some figures on consecutive cases of pulmonary resection with one to two years of follow up. In 48 wedge resections there were no significant complications, 45 of the 48 had sputum conversion. In contrast to these results 18 cases of segmental resection resulted in six failures of sputum conversion, and of these six failures to convert three patients developed serious complications and were made worse by the operation.

In performing the operation of wedge resection it is important to ligate the divided bronchus and vessels carefully with transfixion sutures before the raw tissue is pleuralized. If this is not done one invites the hazards of fistula and bleeding.

DR PAUL T. DE CAMP, New Orleans, La.—I would like to direct attention to the group of patients who have open cavities and positive sputum remaining after adequate chemotherapy. Two years ago, we read before this Association a paper on our initial experiences with a variety of partial thoracoplasty devised by Dr Fernando Paulino of Rio de Janeiro. The procedure is essentially a modified Semb pneumonolysis and partial thoracoplasty. The second rib and parts of the third and fourth ribs are removed. An extra fascial pneumonolysis is performed with division of the intercostal structures. The unique feature of the operation consists in maintaining the pulmonary collapse by placing three ligatures around the diseased lung.

We have had experience with some 45 operations over a four and one-half year period. We are not performing the operation frequently but believe it is often indicated in cases with open cavities and persistent positive sputum because of the increased hazard of resection in these cases. The procedure has the advantage of being a single-stage operation which causes little deformity; the loss of respiratory function is minimal, and the amount of foreign material is negligible. For disease above the hilum the collapse obtained is as good as or better than that following the standard seven rib thoracoplasty. The changes in the collapsed lung are similar to those after the more radical conventional operation.

We have experienced no complications related to the use of ligature constriction of the lung. We have been happy to hear that Dr. James Murphy has used the procedure in a few cases and has been pleased with it. Dr. McLaurin of British Columbia has performed a number of these procedures in the past year and is also satisfied with it. If one has this as an alternative procedure, he may not be tempted to perform a hazardous segmental resection in the face of a positive sputum.

DR RICHMOND DOUGLASS, Ithaca, N. Y. (Closing).—I do think there is a place for some discussion of our terminology. Dr. Lees raised the question of our use of the word "salvage." We could not find a better word. It was applied to our patients for whom there was no other good alternative method of treatment. On the other hand, our elective cases represented a group who had done well by any criteria and one might almost say that the surgery performed was prophylactic in the hope of avoiding relapse. We do feel that it is important to perform this type of surgery in order to have a base line for study and for comparison with patients treated by drugs alone.

In relation to the use of the terms "segmental resection" and "wedge resection" and so forth we have used the term "segmental" when in an anatomic segmental unit of the lung was removed and I think that is perhaps commonly understood. When you get down to small resections there is a multiplicity of terms such as "local excisions," "wedge resections," "block resections" or "subsegmental resections." I think maybe it might be well

to agree on the use of the term "subsegmental" for anything less than a segment, regardless of the technique by which the smaller piece of lung tissue is removed

I wish to thank those who took part in the discussion of this paper

DR ALFRED M DECKER, Summit, N Y (Closing)—I wish to thank the discussants. There are only one or two questions which pertain to our presentation. Dr Overholt brought up a point that has interested us a good deal, and we have had a small experience with early resections. I do believe that so far as lasting tuberculous complications are concerned, resection during the effective period of chemotherapy, that is, the early months, carries very little risk. We have generally practiced delay at least beyond the period of four months because of our gradual evolution of the resection program in prolonged combined chemotherapy. Our experience with the bacteriology of lesions resected has shown that duration of therapy is a factor in this regard. Also we have felt that we were able to perform much smaller resections by waiting for maximal clearing. More recently the major point at issue would seem to be allowing chemotherapy an opportunity to close cavitory lesions. This to our mind, puts the evaluation of further surgical intervention under an entirely different category of disease response to chemotherapy. Our own experience certainly suggests that it remains a very open question whether or not we must resect these residual closed lesions. We are trying to differentiate what we can do from what we should do.

With regard to what Dr Weinberg said about the type of resection affecting the incidence of tuberculous complications, I feel it worth while to make the point that our incidence of tuberculous complications reflected the status of the disease in its response to chemotherapy rather than the type of procedure. Tuberculous complications have been almost nonexistent in those patients who have converted their sputum for periods of three months or longer during continuous combined chemotherapy despite an appreciable incidence of positive cultures in the resected specimens, particularly in those retaining cavitory lesions.

With reference to Dr DeCamp's statements, we certainly do not resect all our chemotherapy failures, and do resort to various adjunctive procedures, particularly, of course, thoracoplasty or thoracoplasty combined with Monaldi in the presence of large cavities. Indications for resection in those patients whose organisms have escaped bacteriologic control entirely have tended to follow the pattern of so called standard indications for resection.

THE ROLE OF PULMONARY INSUFFICIENCY IN MORTALITY AND INVALIDISM FOLLOWING SURGERY FOR PULMONARY TUBERCULOSIS

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BOSTON, MASS

PULMONARY function studies today are generally accepted as an integral part of the evaluation of poor-risk patients who are to have pulmonary surgery. Exertional dyspnea, previous operations or disease on the opposite side are the most common indications. The importance of physiologic evaluation under these circumstances has been emphasized repeatedly.¹⁻⁴ Other reports have dealt with description of techniques and with interpretation of tests. Many new or rediscovered methods have been described for simplified evaluation or for more detailed analysis of pulmonary function. The literature devoted to descriptive reports of pulmonary physiologic alterations in certain disease entities or after specific surgical procedures is constantly increasing. The present analysis was undertaken to assess the value of routine preoperative pulmonary function studies in relation to operative mortality and postoperative disability following surgery for pulmonary tuberculosis in a large unselected group of patients.

PLAN OF STUDY

The observations were begun when the present Thoracic Surgery Service was initiated at the Sanatorium Division of the Boston City Hospital in 1947. Many of the patients awaiting surgery were desperately ill. The considerable number who previously had been refused operative treatment because of clinical dyspnea or far advanced bilateral disease appeared to justify the expenditure of time and energy required for these studies. In addition it was hoped that this program would offer thoracic surgeons in training an opportunity to develop a feeling for and an appreciation of pulmonary physiology.

The laboratory is a division of the Thoracic Surgery Service and all examinations were performed by the surgeons or under their supervision. Preoperative evaluations and follow up studies could thus be closely integrated with the surgical program. This proximity of the laboratory permitted a

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flexible operative program closely guided by physiologic observations. Surgical results were reviewed from time to time in the light of these observations. The surgeons, working both in the operating room and in the laboratory, had an opportunity to observe the physiologic consequences of their operative procedures. Results of function studies were required routine information at weekly surgical grand rounds where problem cases are presented for recommendations.

Both good- and poor-risk subjects were examined for future correlation between the outcome of surgery and the results of initial pulmonary function studies. Only a few simple studies were performed routinely in order that the procedure would not be too difficult for patients and examiners. Thereby it was hoped to continue the project until its value to the surgical program could be assessed. Screening, rather than complete evaluation of the respiratory status was intended. More detailed investigations were initiated when special indications existed (Table I).

TABLE I

ROUTINE PULMONARY FUNCTION STUDIES	
1	Maximal Breathing Capacity (MBC), calculation of per cent of predicted
2	Vital Capacity (VC), calculation of per cent of predicted
3	Timed Vital Capacity, per cent exhaled during 1, 2, and 3 seconds
4	Calculation of Air Velocity Index (AVI) $\% \text{MBC} / \% \text{VC}$
5	Exercise Ventilation, walking 180 feet per minute (WV)
6	Calculation of Walking Dyspnea Index (WI) $100 \times \text{WV} / \text{MBC}$
7	Breath Holding Test (discontinued)
8	Fluoroscopic Observation
9	Bronchoscopic Observation
INDICATIONS FOR SPECIAL STUDIES	
1	Maximal Breathing Capacity very low
2	Timed Vital Capacity low
3	Air Velocity Index less than 0.8
4	Walking Dyspnea Index over 35 per cent
5	Dyspnea not explained by results of function studies
6	Discrepancy between function studies and appearance of roentgenogram
7	Discrepancy between function studies and fluoroscopic observations
8	Surgery planned on the major side (Special Project)
SPECIAL STUDIES	
1	Combined Spirometry (Minute and Tidal Volume, Oxygen Uptake, Ventilation Equivalent Subdivisions of Vital Capacity, Respiratory Pattern)
2	Residual Volume (Total Lung Capacity and RV/TLC ratio)
3	Intrapulmonary Mixing Index (Alveolar N ₂ , per cent after O ₂ Breathing)
4	Arterial Blood Analysis (O ₂ Content and Capacity, CO ₂ content)
5	Bronchospirrometry (Differential per cent O ₂ Uptake Ventilation and VC)

METHOD

Routine studies, carried out within one week of intended operation, are listed in Table I. The planning of this study was aided by communications from Courmand and Richards¹⁰ Ravl,¹¹ Warring,⁴ and Wright.¹² As the study progressed our increasing dissatisfaction with the value of the vital capacity test led to the development of the timed vital capacity and the calculation of the air velocity index.¹⁴ After analysis of the first 1000 routine studies it was demonstrated that the breath holding test has no value in estimating ventilatory function and the test was discontinued. Special efforts were

made to eliminate subjective factors from tests requiring voluntary maximal effort. Such tests as the maximal breathing capacity (MBC) and vital capacity (VC) were repeated, after suitable rest periods until successive efforts checked within 5 per cent. Whenever doubt existed concerning adequate performance, conference pre-entration and operation were delayed until a satisfactory base line had been achieved.

More detailed studies were initiated when special indications existed (Table I). The most frequent additional study was bronchospirometry performed on 312 occasions. Modifications of this technique, developed during the period, have been reported.¹⁷ For measurements of residual volume and intrapulmonary mixing index, the open-circuit oxygen breathing technique was used.¹⁸ All gas volumes are reported at body temperature, ambient pressure and saturated with water (BTPS) with the exception of oxygen uptake which is recorded at standard temperature and pressure dry (STPD). Experiences with ventilatory studies after administration of bronchodilators have been reported.¹⁸ Only volume increase of more than 10 per cent was considered significant. Methods used in this investigation have been discussed in detail elsewhere.¹⁹

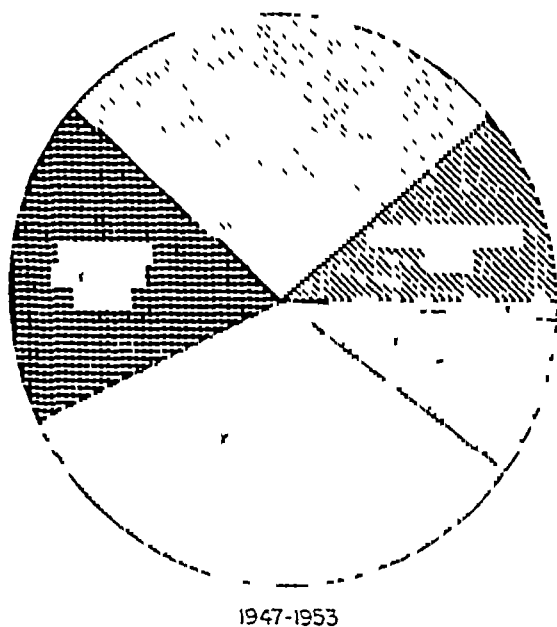


Fig. 1.—Primary surgical procedures. Selection of operations for the treatment of pulmonary tuberculosis for 466 patients during the six years from 1947 to 1953.

RESULTS

Routine studies of 466 patients were made before 839 major operations. The incidence of the type of primary procedures is shown in Fig. 1. The trend in the choice of operations changed during the six year period of observation. During the first two years, staged thoracoplasty was the standard procedure. Major resection during that time was reserved for very definite indications including giant or lower lobe cavity, destroyed lung or bronchostenosis. Extrapleural pneumonolysis was chosen as the most function sparing procedure if pulmonary reserve was very low or if bilateral collapse was planned.^{20,21} During the last two years, extrapleural Lucite sphere plombage replaced conventional staged thoracoplasty almost entirely.²² Lobar and lesser resec-

tions increased in frequency when "localized disease" became one of the indications for resection and when chemotherapy made these operations safer

Respiratory Difficulties Related to Type of Operation—It was anticipated that the type of operation itself, irrespective of the degree of preoperative pulmonary insufficiency, would have a decided effect on the incidence of postoperative mortality and invalidism. Previously, this factor was investigated both during the early²³ and during the later²² postoperative period. Interpretation of these data is difficult because the very choice and extent of surgery are based, to some extent, on the respiratory reserve of the patient. Also a number of unpredictable, function-impairing complications occur only with a certain type of operation. Accidental opening of the pleura with induction of pneumothorax may unexpectedly diminish the respiratory reserve after thoracoplasty, plombage or extrapleural pneumonolysis. Hemothorax, atelectasis or bronchopleural fistula often causes unexpected respiratory difficulties following resection.

Staged thoracoplasty, which accounted for 33 per cent of our operative experience (Fig 1), may serve to illustrate that the extent of operation has marked effect on the diminution of respiratory function regardless of the initial condition of the lung (Fig 2). The MBC decreased about 20 per cent when five or six ribs were removed, but the loss averaged 30 to 40 per cent after more extensive operations. Also typical of staged thoracoplasty, the loss of function early (two weeks) after operation was nearly twice as large as the ultimate diminution six months later. Pain, weakness, and ineffective cough contribute to diminution of respiratory reserve after all thoracic operations. Paradoxical motion of the chest wall adds to the respiratory distress after staged extrapleural thoracoplasty.

Even details of operative technique may have an effect on respiratory mortality, and morbidity. The widely held thought that marginal patients better tolerate thoracoplasty if resection of the first three ribs is performed in two stages guided us early during the program. Consequently, thoracoplasty was initiated by an anterior stage on eleven occasions. Uncontrollable paradoxical motion of the anterior chest wall, accompanied by extreme respiratory distress precluded further surgery in three patients and two died of respiratory insufficiency (Table II). The functional loss of the other eight patients after anterior stage alone was greater than the loss after complete removal of the first three ribs in seventy-three patients (Fig 3). Furthermore, resection of the remaining posterior halves of the three ribs at a second stage was actually accompanied by a gain in function (Fig 3). Suspension at the apex with freedom to move in paradoxical fashion anteriorly appears to be more harmful to function than a collapsed apex confined by pericostal tissues and compressed by fluid. Because of poor results this operative variation was abandoned early but it was in part, responsible for the high (33 per cent) respiratory mortality early after staged thoracoplasty (Fig 4).

After pneumonectomy some degree of respiratory difficulty is anticipated, and in our group, death or some degree of respiratory disability occurred in

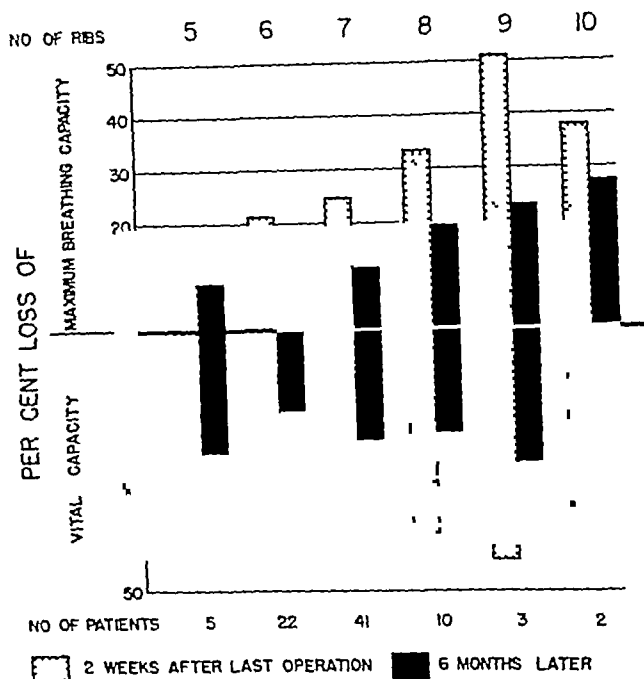


Fig. 2—Ventilatory loss after thoracoplasty. Average per cent decrease of MBC and of VC two weeks and six months after completion of all stages of thoracoplasty arranged according to number of ribs removed. Observations of eighty three patients operated upon from 1947 to 1950.

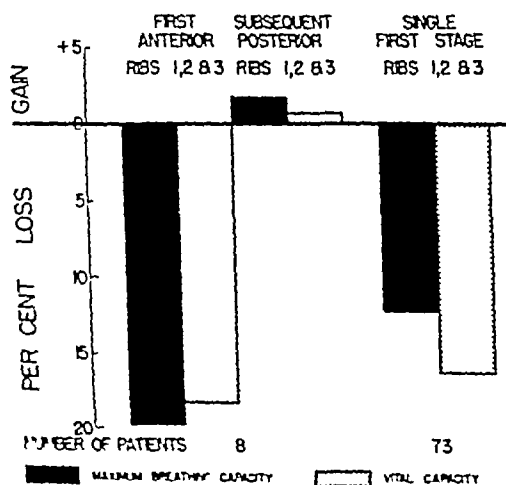


Fig. 3—Loss of pulmonary function after removal of the first three ribs in two stages and in a single stage. Comparison of average per cent decrease of MBC and VC after removal of the first three ribs in eight patients and in a single stage. Removal of only the anterior ribes of the first three ribs in eight patients was followed by a greater loss of function than removal of the entire first three ribs in seventy three other patients.

TABLE II ANALYSIS OF MORTALITY

A OPERATIVE MORTALITY (WITHIN 30 DAYS) 460 PATIENTS, 839 MAJOR OPERATIONS			B LATE MORTALITY (30 DAYS TO 6 YEARS) 440 PATIENTS		
	NUM BER	PER CENT		NUM BER	PER CENT
Respiratory failure*	8	1.7	Respiratory failure (primarily)	6	1.3
Cardiac arrest†	5	1.1	Bronchopleural fistula and empyema*	3	0.7
Operative hemorrhage	4	0.9	Progressive disease	3	0.7
Bronchopleural fistula and empyema	1	0.2	Malnutrition and delirium	2	0.4
Progressive disease or spread	1	0.2	Unrelated to tuberculosis or respiratory failure	3	0.7
Coronary occlusion	1	0.2			
Total	20	4.3	Total	17	3.8

*Associated with progressive pulmonary disease in some patients

†Preceded by technical and respiratory difficulties during anesthesia in four of the five patients

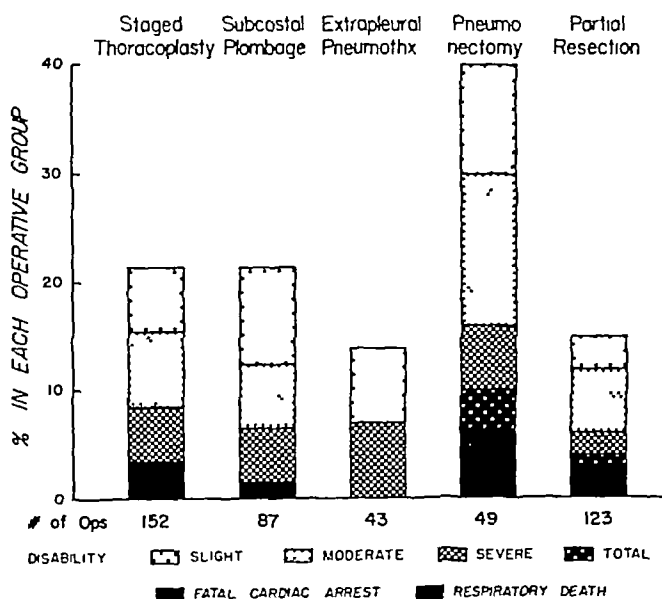


Fig. 4—The incidence of cardiac arrest or death due to respiratory insufficiency and of post-operative disability after various operative procedures

40 per cent. After lesser resections this incidence was only 15 or 20 per cent (Fig. 4). Cardiac arrest occurred only during intrapleural operations. It was responsible for 60 per cent of all operative deaths during pneumonectomy and for 67 per cent of deaths during lobectomy (Fig. 4).

Although extrapleural pneumonolysis was usually reserved for patients with very limited pulmonary reserve there were no operative deaths, and 83 per cent of patients had no subjective respiratory disability six months later (Fig. 4). The function-sparing characteristics of this operation have been discussed elsewhere.

Ventilatory Function Profile of the Entire Group—Points of Fig. 5 indicate the preoperative ventilatory function of every patient with the MBC on

the ordinate and the VC on the abscissa, both expressed as the per cent of the predicted normal. The regression equations for calculation of the predicted normal values have a standard error of about 20 per cent.⁶ Therefore, a performance of 80 per cent or better must be regarded as possibly being within normal limits. Of the whole group of 460 patients 163 or 35 per cent had normal MBC and VC. All patients had at least moderate pulmonary

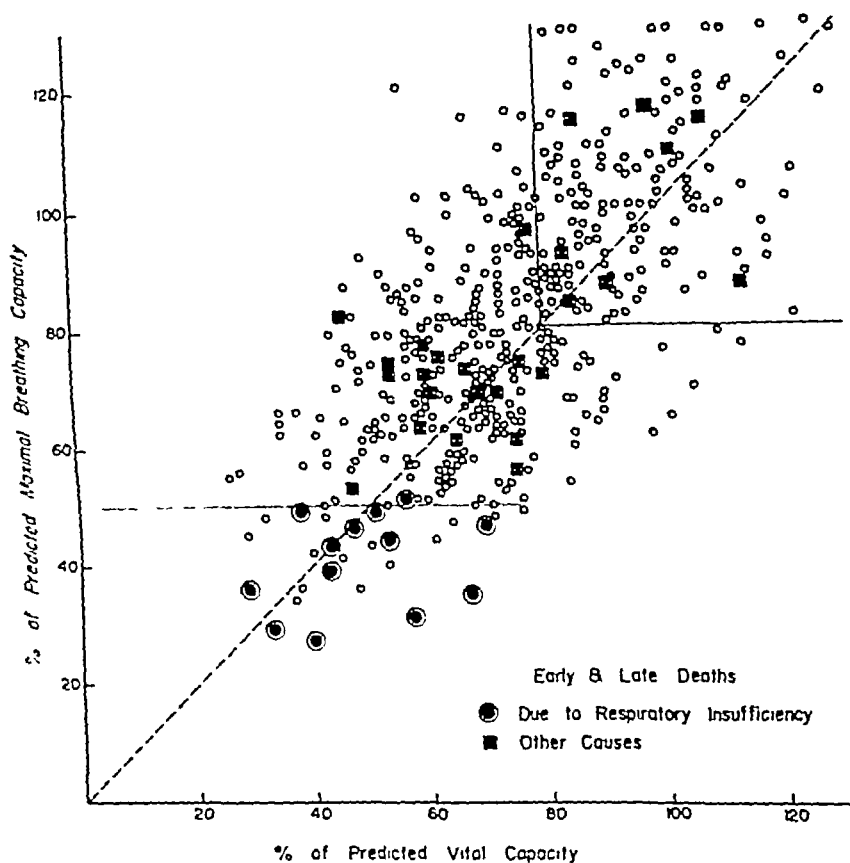


Fig. — Preoperative ventilatory function (460 patients). MBC and VC are indicated as per cent of predicted for each patient. Performance above 80 per cent of predicted was regarded as within normal limits (upper right). The line drawn at forty five degrees to the coordinate axes indicates equal impairment of MBC and of VC (air velocity index = 1.0).

involvement and 75 per cent of the group had far advanced disease. It appears therefore that predominantly unilateral parenchymal tuberculosis per se does not usually impair ventilatory function to a marked degree.

Points falling on a line drawn at forty five degrees to the coordinate axes show equal impairment of MBC and VC. That is the fraction $\frac{\text{per cent of predicted MBC}}{\text{per cent of predicted VC}}$ (air velocity index) is equal to 1.0.¹¹ Most patients had air velocity indices within the normal limits of 0.8 to 1.2 suggesting that a ventilatory defect, if demonstrable, was usually of the restrictive

TABLE III EARLY POSTOPERATIVE DEATHS DUE TO RESPIRATORY INSUFFICIENCY

NO	AGE	SEX	MAI- N CAU- SE OF DEATH	VITAL CAPACITY LITER	AIR FLOW LITER/ MINUTE	WALKING INDEX	BRONCHOSPIROMETRY OPERATED LUNG			PERCENT AT- TAIN- ED	LOS- T OPE- RAT- ION	OPERATIVE PROCEDURE	CONDITION OF OTHER LUNG	COMPLICATING LUNG DISEASE
							% O ₂	% VFN	% VC					
1	11	M	*	*	*	*	*	*	*	3+	4+	Upper lobectomy	7 rib staged thoraco- plasty Emphysema	Bronchopleural fistula, hydro- pneumothorax, "Compensatory," emphysema intrapleural
2	22	M	*	*	*	*	*	*	*	1+	3+	Sulcus ostial plombage		
3	26	F		1.20	1.11	50	31	36	35	0	4+	Anterior stage tho- racoplasty	Extrapleu- ral pneu- mothorax	Severe restrictive pulmonary insufficiency, "Compensatory," emphysema
4	40	M		1.85	0.72	52	38	38	39	2+	4+	Anterior stage tho- racoplasty	Fibrothorax	
5	40	M		1.45	0.91	70	0	0	0	2+	4+	First stage thoraco- plasty	Emphysema	"Compensatory," emphysema, anhydrosis
6	45	F		1.10	1.22	38	0	0	0	2+	3+	Second stage tho- racoplasty	Emphysema	"Compensatory," emphysema
7	48	F		1.18	0.59	69	Tracheostomosis			3+	3+	Second stage tho- racoplasty	Emphysema	"Compensatory," emphysema, tracheostomosis
8	36	M		1.25	1.27	39	0	0	0	0	4+	Decortica- tion	Giant cavity	"Compensatory," emphysema

*Too ill to come to the laboratory

type¹⁴ Only occasional points fall below normal because obstructive ventilatory insufficiency was often masked by more detailed physiologic studies. Reduced air velocity indices were uncommon also because patients with marked emphysema were not usually suitable candidates for surgery.¹⁵

Deaths unrelated to pulmonary insufficiency, indicated by small dark squares in Fig 5 are fairly evenly distributed throughout the chart and occurred in eight patients with initially normal ventilatory function. Deaths primarily due to respiratory insufficiency, indicated by circumscribed dots, occurred with one exception among patients with initial MBC's of 50 per cent of predicted normal or less. A number of patients with such greatly reduced ventilatory capacity survived operation, nevertheless, clinicians who look for a figure conveying a warning against surgery may be impressed that, of twenty eight patients with preoperative MBC of less than one-half of normal 50 per cent did not survive major thoracic surgery. Vital capacities in this group were also greatly reduced but there was no sharp dividing line, and fatalities occurred among patients in whom this volume was 60 and even 70 per cent of normal before operation. Fig 5 shows that, in a group of patients in whom emphysema is not a prominent clinical feature, the correlation between VC and MBC is fairly good if function is not greatly impaired. As MBC decreases, this relationship is almost entirely lost.

Operative Mortality Due to Pulmonary Insufficiency—Deaths were considered operative if they occurred within thirty days after surgery, twenty of the 460 patients or 4.3 per cent died during this period (Table II). In this group pulmonary insufficiency was the primary cause for the fatal outcome on eight occasions or 17 per cent of the cases.

A more detailed analysis of these eight deaths is shown in Table III. Of all 460 patients only two were deemed too ill for transfer to the physiology laboratory for study prior to surgery. Weakness, dyspnea, fever, and excessive cough were the stated contraindications. Both patients (1, 2) died shortly after operation. Therefore it might well be said that patients disabled by chronic pulmonary disease who cannot tolerate the most primitive function test consisting of a trip to the laboratory are not suitable candidates for major chest surgery. This does not apply to patients disabled by acute pulmonary complications when immediate surgery may be lifesaving. Hemorrhage, tension pneumothorax, perforation of the esophagus or chest trauma obviously may prevent pulmonary function studies being performed without precluding operation.

Only two factors were used for inclusion of patients in Table III—death within thirty days after operation and clinically respiratory insufficiency as the primary cause of death. In other respects the group was quite heterogeneous. Ages ranged from 26 to 52 years, the average age of 38 was not significantly greater than that of 36 for the entire group. The disease was extensive and bilateral in some (1, 7), unilateral and complicated by pulmonary emphysema and fibrosis in others (4, 2), and there were several patients with total emphysema (5, 6, 8). Some were not dyspneic during strenuous activity, while others were short of breath during slightest exertion.

Despite their diverse clinical status the patients were surprisingly comparable from a physiologic standpoint. The MBC was less than 45 L or 50 per cent of predicted in all. All but one had a VC below 15 L. In all members of the group, the walking index was above the "dyspnea level" of 35 per cent,⁴ and four of the eight required more than 50 per cent of their maximal ventilatory capacity for this leisurely exercise.

Respiratory embarrassment may be anticipated if operation is performed on the major side. Split function studies were obtained in all patients in whom this situation was suspected (Table II). On ninety-six occasions patients were actually operated upon on the side responsible for more than half of the respiratory work. Unexpectedly, none of these died of respiratory insufficiency. On the contrary, three respiratory deaths occurred after operation on a lung known to be entirely devoid of function (5,6,8), and a similar situation probably existed in one other who did not have bronchspirometry (7). A procedure performed on a nonfunctioning side may thus have a decided effect and occasionally a catastrophic one on the respiratory status. If pulmonary reserve is minimal and the major lung is emphysematous, anesthesia alone may upset the delicate balance of borderline chronic hypercapnia. The respiratory center, depressed by excessive carbon dioxide, may never again regain control. The high oxygen-gas mixtures customarily administered at these times only aggravate the condition (5). Permanent deterioration of function may occur after thoracoplasty upon a nonfunctioning lung."

Pulmonary emphysema of the so called "compensatory" type was the single most important contributor to preoperative respiratory insufficiency. Emphysema could be demonstrated clinically, roentgenographically, or by lung volume and intrapulmonary mixing studies in all but one patient of this group (3). In spite of this, a very low air velocity index was demonstrable in only two patients. In the others the obstructive defect was masked by severe restriction of the lung volume due to parenchymal or pleural disease.

The severity of dyspnea is often considered for clinical evaluation before surgery. Statements concerning shortness of breath may be very misleading because one has to rely upon subjective consciousness of a sensation which is difficult to describe. After pulmonary disease of long standing, an awareness of undue effort on breathing may disappear and dyspnea denied even while labored breathing is obvious to the examiner. Lack of opportunity to experience dyspnea due to prolonged bed rest, failure to distinguish between dyspnea and muscular fatigue^{19, 28} and purposely misleading statements concerning dyspnea in order "to be accepted for surgery" all contribute to the unreliability of this symptom in preoperative evaluation. Indeed, only two of the eight patients who died early of respiratory failure admitted to more than slight shortness of breath before surgery (Table III). Equally disturbing and more common are the cases where surgery is denied because of dyspnea and where laboratory examination later shows adequate pulmonary reserve.

Nearly all operative deaths occurred after staged thoracoplasty. This is not necessarily a reflection on the operation. Other patients with almost equally poor function survived staged thoracoplasty without great difficulty.

(Fig. 5) Furthermore, patients with initial pulmonary function as low as those shown in Table III were never selected for resection. Initially, patients with poor function were deliberately chosen for thoracoplasty because of the possibility of staging with variable time intervals depending on functional recovery. Three patients never improved sufficiently to have a second operation so that the advantage of staging did not apply. In two others the first stage was followed by a stormy course with extreme dyspnea, cyanosis, and in one case with coma. Both died after the second stage although a much longer interval than the usual two weeks was allowed between operations. In other cases, variable intervals between stages, depending upon recovery of function, proved to be advantageous. With increasing experience it was found that subcostal extraperiosteal plombage is more easily tolerated during the early postoperative period than even the first stage of thoracoplasty with its unavoidable paradoxical motion of the chest wall²⁰⁻²². This does not apply if the pleura is accidentally opened at operation. The function loss due to these two procedures is more comparable when comparison is made some months after operation.

Late Deaths Due to Respiratory Insufficiency—Of the 440 patients who completed the surgical program seventeen or 3.8 per cent died more than thirty days after operation. Discussion will be confined to the six patients (1.3 per cent) who died primarily of respiratory failure although pulmonary insufficiency was a contributory cause in the deaths primarily due to progressive disease, bronchial fistula, empyema, malnutrition, or alcoholism (Table II). Clinical data of the six patients are shown in Table IV. The function studies of the first two patients were obtained only fourteen days postoperatively because death occurred within six months. The remaining follow-up studies were made six months or more after operation.

The entire group has little in common. On physiologic and clinical grounds the six patients may well be discussed in pairs. The first two patients had adequate function initially. One (9) performed better than the mean for the group and the other (10) had better function than the patients considered to have marginal pulmonary reserve. Neither patient admitted dyspnea. In both instances the disease was predominantly unilateral and far advanced and repeated pulmonary hemorrhage was the most alarming symptom. An eight rib staged thoracoplasty was performed on the minor side of both patients but failed to control bleeding. On the contrary, with increasingly labored breathing after successive stages these episodes became more frequent and more severe. Marked paradoxical chest wall motion prevented adequate elimination of blood and secretions. The much reduced pulmonary reserve after operations was not only the result of extensive thoracoplasty but was also due to retained secretions, pneumonitis, and atelectasis. These complications were eventually responsible for death due to respiratory insufficiency.

The prominent clinical feature in the second two patients (11-12) was a history of severe bronchial asthma since childhood occurring seasonally but also elicited by emotional stress. Very severe pulmonary insufficiency, ob-

TABLE IV LATI POSTOPERATIVE DEATHS DUE TO PULMONARY INSUFFICIENCY

NO	AGE	SEX	MAXI- MUM BREATH- ING CAP- ACITY LITERS/ MINUTES	VITAL CAPACITY LITER	AIR VENT- ILATION INDEX	WALK- ING DYS- PNOEA INDEX	BRONCHOSCOPY OPERATED LUNG			PROF- FUSIVE DYS- PNOEA	POST- OPER- ATIVE DYS- PNOEA	OPI- RATORIAL PROCEDURE	CONDITION OF OTHER LUNG	COMPLICATING PULMONARY DISORDER
							% O ₂	% VENT- ILATION	% VENT- ILATION					
9	19	M	93 31	1.29 1.10	0.80 0.63	26 68	40 —	44 —	41 —	1+	4+	9 rib staged thoraco- plasty	Minimal disease	"Compensatory," emphysema, pulmonary hemorrhage
10	35	M	67 10	1.95 1.21	1.15 1.10	38 63	36 —	39 —	38 —	0	4+	8 rib staged thoraco- plasty	Progressive disease	"Compensatory," emphysema, pulmonary hemorrhage
11	20	F	49 71	1.31 0.81	0.93 1.14	47 60	76 —	73 —	64 —	Var	Var	Phrenic, pneumo- perito- neum, cavernos- tomy	Extrapleu- ral pneu- mothorax	Bronchial asthma, cor pulmonale, cardiac failure
12	18	F	71 10	1.78 1.01	0.95 1.23	42 56	26 0	34 3	32 0	Var	Var	9 rib staged thoraco- plasty	App nor- mal	Bronchial asthma, status asthmati- cus
13	50	M	10 31	1.70 1.38	0.86 0.80	41 51	96 —	69 —	72 —	1+	3+	5 rib sub- costal plombage	10 rib total thoraco- plasty	"Compensatory," emphysema, cor pulmonale, car- diac failure
14	33	M	67 11	2.29 1.89	0.93 0.92	32 40	— —	— —	— —	2+	4+	7 rib sub- costal plombage	Pulmonary fibrosis	"Compensatory," emphysema, cor pulmonale, car- diac failure

Figures in Italics are postoperative data

served initially in both patients was due to advanced tuberculosis pneumo-peritoneum phrenic paralysis, cavernostomy, and intrapleural pneumothorax in one and therapeutic pneumothorax complicated by atelectasis in the other (Fig. 6). A moderate degree of obstructive emphysema was demonstrated in both and this was not reversed by inhalation of bronchodilators. Both patients were free from asthma during their stay in the hospital. Operations were tolerated without unexpected respiratory distress. At the time of discharge both were apparently well in spite of severely reduced function (Table IV). The first patient (II) was readmitted one year later with severe bronchial asthma and rapidly progressive cor pulmonale with massive ascites

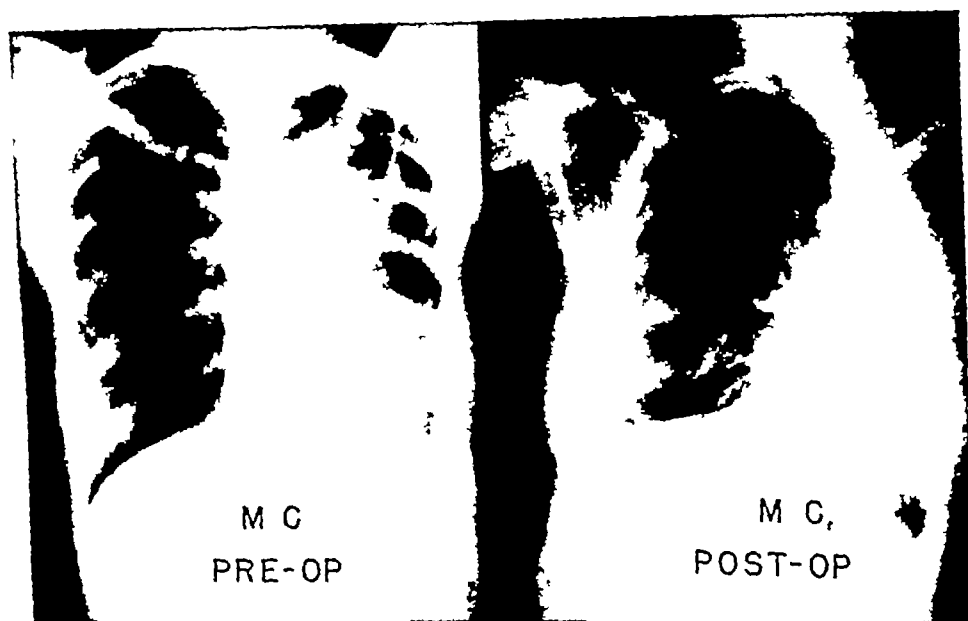


Fig. 7.—Patient 12 gave a history of bronchial asthma. The preoperative roentgenogram shows extensive parenchymal disease on the left side with an unsatisfactory result after therapeutic pneumothorax. The late postoperative film shows marked scoliosis after an eight-rib thoracoplasty.

Death was due to combined respiratory and cardiac failure. Physiologic data from this woman have been reported in detail.²² The asthmatic attacks of the other patient recurred as soon as she returned to her former environment (Fig. 6). She was seen at the laboratory on several occasions with severe respiratory distress and demonstrable hypoxemia and hypereapnia. On these occasions ventilation improved after bronchodilators. This patient died at home with what was described by her physician as status asthmaticus. In both cases the course of bronchial asthma was apparently accelerated by the added cardiorespiratory burden imposed by tuberculosis and by collapse therapy.

The last two patients were in an older age group. Moderate pulmonary emphysema could be demonstrated in both. An old ten rib thoracoplasty con-

tributed to the pulmonary insufficiency in one case (13, Fig 7). Marked pulmonary fibrosis was responsible for hyperventilation and severe disability in spite of a fairly adequate ventilatory capacity in the other (14). A history of cardiac failure and signs of cor pulmonale served as a warning in both patients. Because of this, extrapleural pneumonolysis was selected as the most conservative procedure. However, the extrapleural spaces could not be maintained, apparently because the rigidity of the lungs prevented collapse by an pressure. A small subcostal plombage was then resorted to and resulted in sputum conversion in both patients (Fig 7). A few months after they left the hospital, cardiac failure recurred. Neither patient responded to the usual measures. Oxygen therapy, instituted because of increasing dyspnea at rest, resulted in hypoventilation and hypercapnia in one patient and was of no benefit in the other. Both died in less than one year after operation.

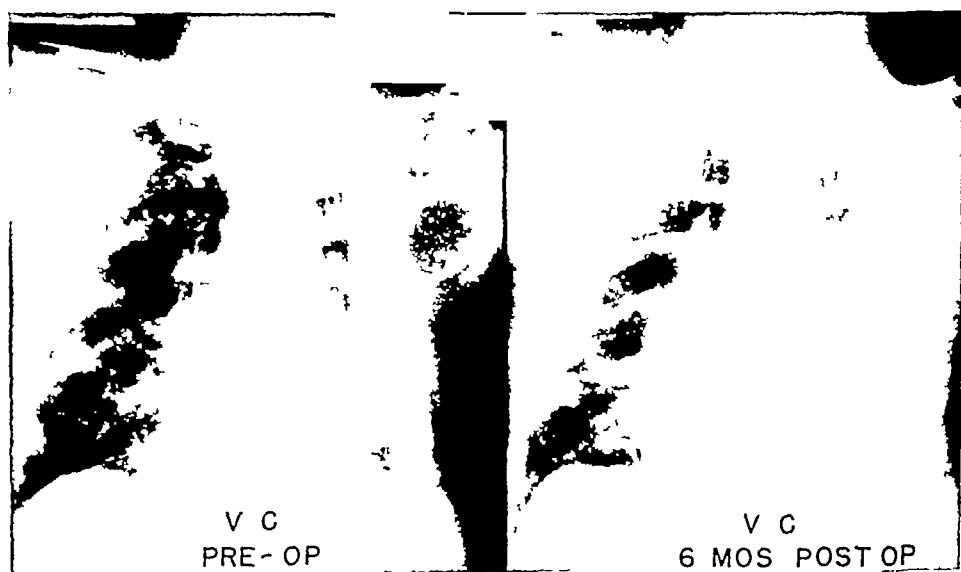


Fig 7—The preoperative roentgenogram of Patient 13 was obtained ten years after a ten rib staged thoracoplasty had been performed on the left because of empyema and four months after extrapleural pneumonolysis had been attempted on the right for treatment of an apical cavity. The disease was arrested by subcostal plombage.

Postoperative Disability—The majority of patients who have had surgery resulting in arrest of pulmonary tuberculosis return to a normal life and useful activity. A somewhat altered mode of life and a more sedentary occupation are often advised for prevention of relapse. Occasionally, inactivity, unemployability, and invalidism can be traced to respiratory insufficiency manifested by dyspnea. In patients with chronic pulmonary disease "disability" and "dyspnea" are to all intents synonymous. The subsequent disability (or dyspnea) was graded in the following manner: it was termed "slight" if subjective symptoms appeared with unusual exertion; "moderate" if they occurred while the patient was climbing one flight of stairs; "severe" dyspnea was noted while the patient was walking on a level surface at a leisurely rate,

disability was "total" if there were symptoms at rest or with slightest exertion. Postoperative evaluation of dyspnea was made at least six months after operation when there had been an opportunity to test limitation of activity with graded exercise.

Subjective symptoms of dyspnea have been correlated with results of pulmonary function studies in a number of papers already referred to.^{14, 15, 26, 28} For the present analysis *postoperative* respiratory disability was related to (1) type of operation, (2) degree of *preoperative* respiratory disability, and (3) certain *preoperative* pulmonary function studies.

(1) The incidence of dyspnea and disability was definitely related to the type of operation (Fig. 4). Of the patients who were well after thoracoplasty, 18 per cent reported subjective symptoms of respiratory insufficiency. However, none were totally disabled and in only 5 per cent was dyspnea severe enough to prevent useful activity.

Subcostal plombage was less prone to cause dyspnea during the early postoperative period but at the end of six months the incidence of disability was almost identical to that staged thoracoplasty. Over the course of years staged thoracoplasty may be more harmful to function than subcostal plombage which does not require resection of transverse processes and of the first rib. The thoracic deformity and scoliosis after resection of these structures appear to impair function of the opposite lung and may favor the development of compensatory emphysema.^{16, 20, 22, 24, 27, 30}

Intrapleural pneumothorax was established and maintained with minimal additional respiratory distress in spite of the fact that most patients had had previous contralateral surgery and marginal function preoperatively.^{20, 21} Six months after operation none were totally disabled and only 14 per cent noted any dyspnea (Fig. 4).

Disability was greatest after pneumonectomy: more than one-third of the patients had respiratory symptoms. Among the survivors 10 per cent could not engage in any useful activity and 4 per cent reported dyspnea at rest or with slightest exertion. Less than two thirds of the group were living and free of symptoms six months to six years later.

Partial resection was rarely followed by respiratory symptoms particularly if only a segment or wedge was removed. Individual variation of postoperative pulmonary function and disability was greatest in this group, and unexpected end results due to complications were more frequent than after other types of surgery.

(2) Postoperative dyspnea was intimately related to the severity of dyspnea before operation. This relationship held even when type of operation and complications were disregarded (Fig. 8). Slight postoperative dyspnea was present in thirty-three patients or 72 per cent of the entire group. More than two thirds of them had been equally short of breath before surgery and only two were improved afterward. Moderate postoperative dyspnea was reported by thirty-five patients or 76 per cent of whom one half had been equally disabled before operation. In this group only two patients had been free from respiratory distress preoperatively and only two felt better after surgery.

Severe postoperative dyspnea was found in twenty-four patients or 5.2 per cent all of whom had been dyspneic before surgery. The eleven patients or 2.4 per cent with the most severe postoperative dyspnea had reported all degrees of shortness of breath before operation (Fig 8, Tables III and IV). More significant was the fact that nine of these eleven patients died within one year after surgery. Apparently pulmonary insufficiency severe enough to cause total disability is not long compatible with life.

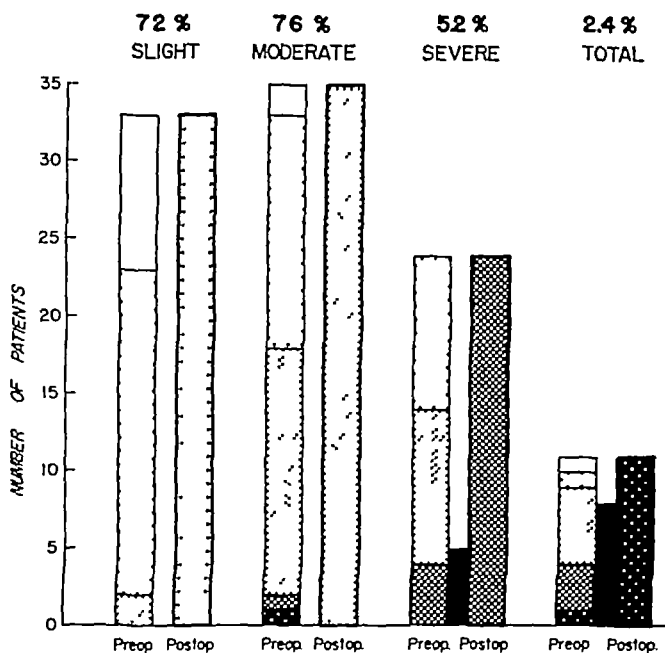


Fig 8—The relationship of late postoperative disability or dyspnea to disability before operation (460 patients). Postoperative deaths due to respiratory insufficiency are indicated by the central columns in black. The code is the same as in Fig 4.

(3) Correlation between postoperative disability and preoperative function studies is less relevant because important variables introduced during the interval are neglected. The analysis was undertaken, however, to determine whether any relationship exists between initial function and later disability, irrespective of the choice of operation and intervening complications. Patients who died of respiratory insufficiency were included in Figs 9 and 10 as the extreme of disability. An almost linear relationship existed between preoperative MBC and late postoperative disability. Patients who later had no symptoms of insufficient pulmonary reserve had a mean preoperative MBC at 81 per cent of predicted. With increasing postoperative disability the preoperative MBCs were smaller. The mean was only 48 per cent for those totally disabled after surgery and 38 per cent of normal among the respiratory fatalities. Fig 9 re-emphasizes that, irrespective of type of operation or complications, an initial MBC of less than 50 per cent should serve as a warning: the majority of patients in this category did not survive or were very severely

to totally disabled by operation. The vital capacity paralleled the MBC at first. With increasing disability this relationship was lost. The average vital capacities for patients in the moderate, severe, and total disability groups were all the same. A progressive increase of the air velocity index was the result.

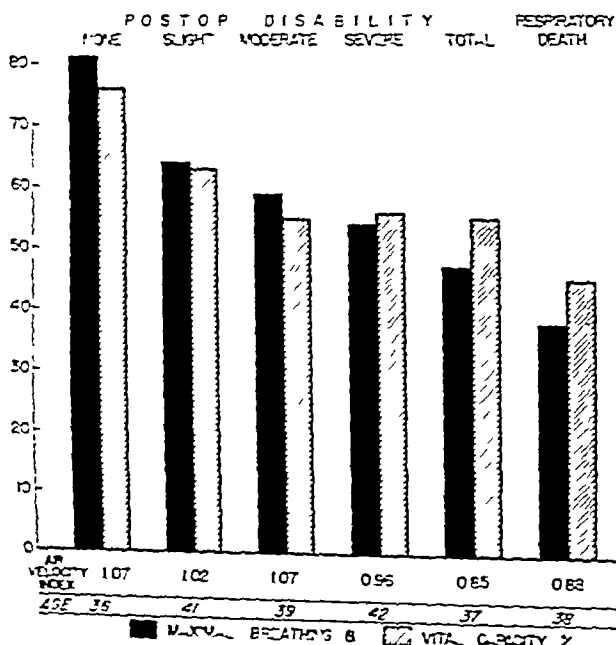


FIG. 2.—Average postoperative MBC and VC as per cent of predicted, arranged according to the degree of late postoperative disability.

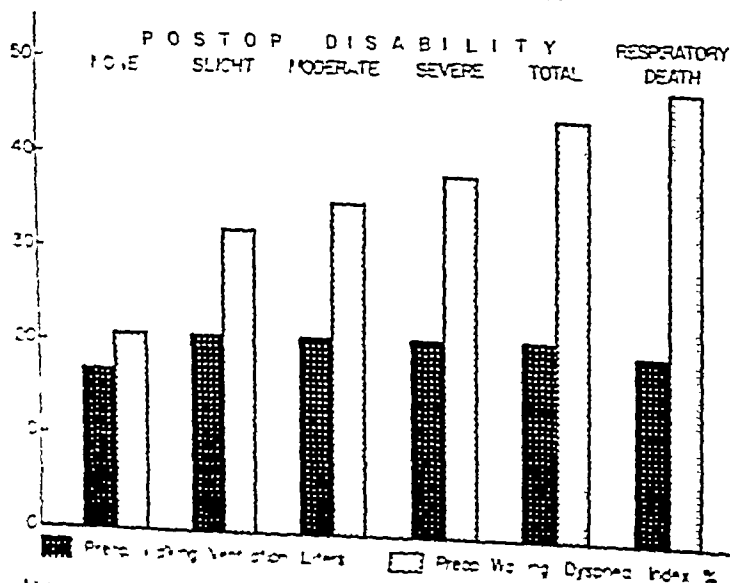


FIG. 3.—Average preoperative ventilation index and walking speed in 10 minutes, arranged according to the degree of late postoperative disability.

Exercise ventilation was of no prognostic value no matter whether this was expressed in liters per minute or in liters per square meter of body surface area (Fig 10). The average walking ventilation of 18 L per minute among relatively normal patients increased slightly to 20 L per minute among those who later complained of slight dyspnea. The ventilatory requirement for this standard exercise was then the same, irrespective of the degree of postoperative disability. Those who later died of respiratory insufficiency actually showed a slight reduction of walking ventilatory requirement (Fig 10). This was probably an expression of underventilation: the pulmonary reserve was so limited that the body could no longer adequately respond to the stimulus. Hyperventilation during mild exercise is thus not a prominent feature and should not be considered of diagnostic significance. Among the tuberculous patients who consistently do show a trend toward hyperventilation are those with pleural fibrosis, fibrothorax, or empyema.³¹⁻³³ This is probably due to increased reflex stimulation of the respiratory center. Impaired oxygen diffusion capacity leads to demonstrable hyperventilation only during the acute phase of hematogenous pulmonary tuberculosis.³³

The walking index or ratio of exercise ventilatory requirement to maximal ventilatory capacity (MBC), increased progressively as postoperative disability became more prominent. Because the numerator of the fraction remained essentially unchanged, the walking dyspnea index was entirely a reflection of the maximal breathing.

Improvement in pulmonary function and a decrease of dyspnea after collapse therapy or resection have been reported, such cases are frequently shown at therapy conferences. From Fig 8 it appears that a decrease in dyspnea after operation was reported by only three patients. Detailed physiologic studies lead us to believe that a gain in function after such operations is extremely rare. If it does occur this finding is usually readily explained by reversion of previously atelectatic lung, resolution of pneumonic infiltration or resorption of intrapleural air or fluid. Very occasionally general debility, weakness, and excessive sputum preclude maximal effort before operation. In such patients the elimination of secretions and recovery of strength after surgery may give rise to an apparent improvement of function which overshadows the actual loss due to surgery. With preoperative preparation with presently available chemotherapeutic agents such cases should be rare. It has been said that reduction in the size of the hemithorax should benefit patients with obstructive or "compensatory" emphysema. We have never seen such a patient's respiratory status improved by any form of collapse therapy except pneumoperitoneum. On the contrary, these are the patients who are most vulnerable to any interference with the bellows mechanism of thorax and diaphragm (Tables III and IV).

CASE HISTORIES

CASE 7.—W. McG., a 36-year-old machinist, was first hospitalized in 1941 because of bilateral pulmonary tuberculous infiltration with cavities at the apex of the right lung. Treatment with artificial pneumothorax and closed pneumonolysis was followed by persistent pleural effusion. When pneumothorax refills were abandoned because of total

atelectasis, he left the Sanatorium against advice, and after resting at home for eighteen months, he returned to work. During 1946 he was briefly readmitted because of hemoptysis. A chest roentgenogram showed a homogeneous density over the entire right lung field. Once more the patient left contrary to advice. At the time of his last admission in 1947, he was cachectic with severe clubbing of the fingers. The chest roentgenogram showed hydropneumothorax with total atelectasis on the right. There was a 100 per cent Congo red retention. Purulent tuberculous drainage continued for three years after dependent closed thoracotomy. Despite suction and prolonged antimicrobial therapy, there was no improvement (Fig 11). At this time there was very severe ventilatory insufficiency with MBC 35 L per minute (29 per cent), VC 1,450 cc (32 per cent), and walking dyspnea index 70 per cent (Table III). Bronchspirometry revealed the right lung to be functionless. Operation was undertaken after considerable deliberation over the course of one year. The decision to operate was influenced partly by the patient's desire for surgery and partly by the feeling that collapse on the side entirely devoid of function should not result in further respiratory impairment. The patient denied dyspnea with moderate activity although doctors and nurses had observed breathlessness even at rest.

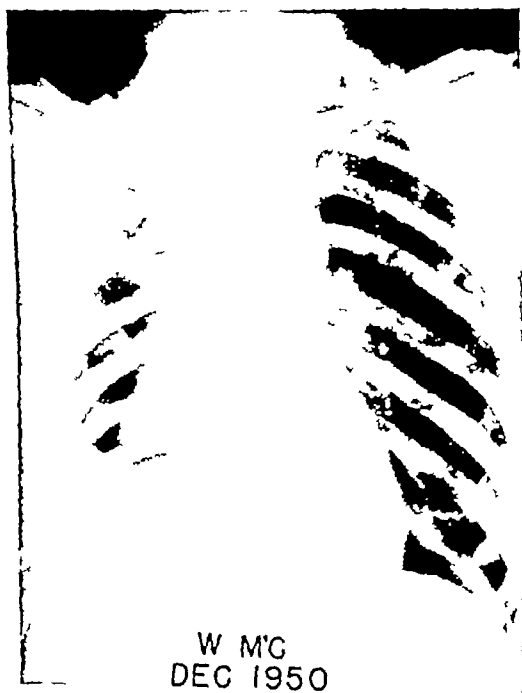


Fig 11—Preoperative chest roentgenogram of Patient 5. A total tuberculous empyema on the right had been drained dependently. The right lung was functionless. Severe contralateral emphysema was demonstrated.

Stage I thoracoplasty under local anesthesia was begun by resection of the first three ribs in an attempt to close the emphysema space. Early during the operation the patient complained of shortness of breath and oxygen was given by mask. A few minutes later there was a fall of consciousness and cessation of spontaneous respiration with a moderate fall in blood pressure. The episode was attributed to acute respiratory acidosis and carbon dioxide narcosis. The patient was intubated and artificial ventilation by intermittent positive pressure was instituted. The blood pressure returned to preoperative values after transfusion of 100 cc of blood. At the end of operation the patient regained consciousness and the detached chest tube was removed. However, spontaneous respiration failed

to resume, and the patient breathed only when urged to do so. Because of cyanosis, not accompanied by any urge to breathe, electrophrenic stimulation was instituted and continued for six hours. Fairly adequate respiratory excursions then resumed, but breathing remained labored. In spite of long standing emphysema and markedly thickened parietes (Fig 11) there was a marked mediastinal swing and paradoxical motion of the anterior chest wall. During the first two days after operation, oxygen was given intermittently because of cyanosis and dyspnea. This was always followed by diminution or cessation of respiratory excursions. Ventilation became progressively less effective, and the patient died on the fourth day after operation.

CASE 7—M. R., a 38 year old housewife, was first admitted in 1939 with bilateral apical infiltration and a cavity at the apex of the right lung. She was discharged after three years of bed rest treatment, the disease apparently arrested. She remained well for eight years but was then readmitted because of marked weight loss and a productive cough. The chest roentgenogram revealed extensive cavitation at the apex of the right lung. The clinical response with pneumoperitoneum, streptomycin, and PAS was poor. Six months after admission there was sudden onset of dyspnea, fever, and chills, and the roentgenogram revealed complete obliteration of the right lung field. Bronchoscopically, severe endotracheal and bronchial ulcerations were observed, and there was a stenosis of the trachea.



Fig 12—Preoperative chest roentgenogram of Patient 7. Bronchoscopic observation revealed a low tracheostenosis with obstruction of the right main bronchus. Marked compensatory emphysema was demonstrated.

with complete obliteration of the right main bronchus. Further prolonged treatment with chemotherapy including isoniazid resulted in improvement of tracheal lesions but the complete obstruction of the right main bronchus persisted (Fig 12). Surgery was considered as a last resort in an attempt to reverse the progressively downhill course. At that time the MBC was 22 L. per minute (33 per cent), the VC 1,480 (47 per cent), and the walking dyspnea index was 60 per cent (Table III). The right lung was presumed to be functionless, but bronchospirrometry was not performed because of the conditions of the major airways. Following a first stage thoracoplasty in April, 1953, secretions became difficult

to raise. There was a harassing cough and severe dyspnea. The patient eventually improved sufficiently for a second stage with resection of the fourth and part of the fifth ribs to be performed one month later. Immediately afterward breathing became labored, and there was marked paradoxical motion of the chest wall. In spite of intermittent oxygen therapy, endotracheal suction, and other supportive measures, dyspnea became progressively more severe and cough less effective. On the second postoperative day, following a paroxysm of coughing, the patient became deeply cyanotic and lapsed into coma. In spite of endotracheal oxygen insufflation, she died a short time later.

CONCLUSIONS AND SUMMARY

Screening studies of pulmonary functions were performed before operation in 460 patients with advanced pulmonary tuberculosis.

Early mortality was 4.3 per cent. Respiratory failure accounting for eight of the twenty deaths during this period, was the most important cause of early mortality. All eight patients in this group had a preoperative MBC of less than 45 L. per minute, a vital capacity of less than 2 L., and a walking dyspnea index of over 35 per cent. "Compensatory" emphysema was the most important factor contributing to pulmonary insufficiency and was demonstrated in all but one of the patients who died of respiratory failure. The preoperative function of the lung to be operated upon was not a factor in mortality; in four of the eight patients who died, the involved lung had been devoid of function preoperatively. Among ninety-six patients who had surgery on the major side, there were no fatalities.

Late mortality was 3.9 per cent. Respiratory failure was responsible for six of the seventeen deaths which occurred from thirty days to six years after operation. Recurrent bronchial asthma, cor pulmonale with cardiac failure, and 'compensatory' emphysema were the main complications in this group.

Some dyspnea was reported after recovery by 22.6 per cent of the patients, and this led to severe or total disability in 7.8 per cent. Pulmonary insufficiency, severe enough to cause dyspnea at rest or with slightest exertion, was not compatible with life for more than a few months. Postoperative disability could be related to the type and extent of operation, to the degree of preoperative dyspnea, and to objective measures of preoperative pulmonary function.

The access to objective data concerning pulmonary function, at the time operative decisions were made, did not eliminate entirely postoperative invalidism and deaths due to respiratory failure.

Most often, an unfortunate outcome after surgery occurred when pleas of patient, relatives, and ward physician prevailed over objective data from the laboratory. A number of poor results were due to the misconception of the cry that staged thoracoplasty, particularly anteriorly, is more easily tolerated than other collapse procedures. Function studies were of little prognostic value in patients with bronchial asthma if evaluation was made during the symptomatic phase. Two patients who were considered to be too ill to have function studies did not survive operation. Unexpected disability after recovery usually could be traced to operative complications. Accidentally

induced pneumothorax, hemothorax, atelectasis, bronchopleural fistula with empyema, and extension of the disease were the most frequent offenders in this regard

Rarely misinterpretation of available data was responsible for respiratory fatalities or undue disability after operation. The MBC and other tests which are sensitive to obstructive ventilatory insufficiency, particularly timed vital capacity and air velocity index, proved of greatest prognostic value. The VC was often misleading and was useful chiefly when viewed in the light of other function tests. The walking dyspnea index usually was merely a reflection of the MBC and not of exercise ventilatory requirement. The breath holding test proved to be worthless. Bronchspirometry was the most helpful of the special tests. Residual volume and intrapulmonary mixing determinations in these cases served chiefly to differentiate true emphysema from hyperinflation. With increasing experience the interpretation of these tests improved and results were viewed with greater confidence. Thirteen of the fourteen "respiratory" deaths occurred during the first three of our six-year experience.

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DISCUSSION

DR. CLAIR BASINGER, Chicago, Ill.—In the absence of Dr. William Adams he has suggested that I present some of our experiences in the problem of pulmonary insufficiency. It has been our feeling that pulmonary hypertension is a significant factor in the death of these patients with reduced pulmonary reserve. Data presented before this Association one year ago by Dr. Adams showed that dogs died of heart strain when the pulmonary artery pressure was increased 100 per cent or more than normal values in animals in which the pulmonary function was reduced to 15 per cent of normal. Experiences over the past year in four patients with extreme reduction in pulmonary reserve as measured by accepted pulmonary function studies have shown that in three cases there was objective evidence of heart strain and/or decompensation before or after operation as measured by increased pulmonary artery pressure or signs of decompensation. The recording oximeter was useful in managing postoperative therapy following intrathoracic surgery.

(Slide.) This is a 55-year-old man with carcinoma of the right lung, and the data are from the oxygen saturation twenty-four hours postoperatively. With 2.5 to 5 L. of oxygen the saturation was brought up to 94 to 96 per cent. In checking these patients the one finding has been that seldom is more than 5 L. of oxygen necessary to bring peripheral arterial oxygen up to normal values.

(Slide.) This is a 64-year-old man with markedly reduced pulmonary function, a carcinoma of the right lung was found and a right pneumectomy was performed. He did well for the first ten days following operation but then increasing amounts of oxygen were required. He died twenty days following operation from cor pulmonale and right heart failure. This was the mean preoperative pulmonary artery pressure 31 cm. of saline solution, and the level of the pulmonary artery. Postoperatively after ligation of the artery the level fell to 7 mm. of this solution. The normal mean pulmonary artery pressure is 15 to 20 mm. of saline solution. It is felt that this man then had about 105 per cent increase in this pressure.

These data bring out the role of pulmonary hypertension as a factor in heart strain, perhaps contributing considerably to the death of these individuals. Relatively small amounts (1 to 2 L/min) of oxygen are necessary to increase the oxygen saturation values and bring them to normal. From these experiences it would appear that oximetry would be helpful in estimating the risk of resection in patients with severely reduced pulmonary function. It is realized that the cases spoken of are not pulmonary tuberculosis, but we think the principles are equally applicable.

DR PAUL NEMIR, Jr, Philadelphia, Pa.—I have greatly enjoyed Dr Gaensler's fine presentation. We have been interested, also, in finding methods of obtaining more accurate preoperative evaluation of pulmonary function in the poor risk patient or the patient with borderline pulmonary reserve. About two and one half years ago, we began studying pulmonary function by using a method originally devised by Carlens, Hanson, and others in Crafoord's clinic, wherein controlled occlusion of the right or left pulmonary artery and the corresponding bronchus was carried out in the intact unanesthetized patient. We have been using a special triple lumen cardiac catheter, with recording lumina both proximal and distal to an occluding balloon. This catheter is passed in routine fashion into the right or left pulmonary artery, and the balloon is inflated with Diodrast until the artery is occluded. A Carlens endotracheal catheter has been used to occlude the bronchus on the corresponding side. In this manner, we feel we have created for all practical purposes a physiologic "pneumonectomy" and studies have been made on the function of the remaining lung. Through the triple lumen catheter, we have been able to obtain pressures at various locations in the pulmonary system and, in addition, careful gas analysis studies have been performed. Our studies have confirmed many of the observations which the essayist has related. We have studied about twenty three patients at present, and almost all have been poor risk patients or patients with borderline pulmonary reserve as determined by the ordinarily used methods. The small number of course, allows no definitive conclusions, however, there has been a definite relationship between a sustained increment of the main pulmonary artery pressure following occlusion of its right or left branch and the occurrence of dyspnea in the post-operative period following resection. We have felt that this will be a good method for determining resection tolerance in poor risk patients undergoing extensive pulmonary surgery.

DR WARREN WOODRUFF, Saranac Lake, N. Y.—Dr Gaensler's emphasis on the importance of pulmonary insufficiency as a cause of mortality and invalidism is very timely. I believe he would agree that studies of pulmonary function are being used increasingly more but that many patients with borderline function are still being subjected to surgery without the advantages of such information. It is true that most patients will withstand major thoracic surgery without incident because we are normally endowed with great pulmonary reserves. In the poor risk patient however, it becomes obligatory to have accurate knowledge of the extent of impairment.

I should like to ask Dr Gaensler if early in his studies he did not find that his contemplated surgical approach had to be changed because of his findings. We have had that experience on a number of occasions.

I agree with Dr Gaensler that a Lucite thoracoplasty is frequently better tolerated than a routine thoracoplasty or a resection but it should be emphasized that the reason is that with this method it is possible to maintain an intact thoracic cage which is particularly important in a patient with limited reserves.

I am not clear as to the importance he attaches to vital capacity. We have felt for a long time that the vital capacity, of itself is of little value and in many instances has been misleading.

DR EDWARD A. GAFNSIER, Boston, Mass. (closing).—Dr Baesinger's comments are borne out by our clinical observations. In most of our late respiratory deaths pulmonary hypertension (or pulmonary and cardiac failure) were the important complications. The lungs have a tremendous capacity for handling increased blood flow without appreciable rise in blood pressure. Most of our patients had unilateral or well localized disease and pulmonary hyper-

tension was not demonstrable. When there was an additional diffuse process, usually emphysema, asthma, or pleural disease, increased resistance to blood flow could be demonstrated at times. Chronic emphysema and fibrothorax were the worst offenders in diminishing the lung's ability to handle increased blood flow, during bronchospirometry the oxygen uptake was always much more severely diminished than ventilation.

Ear lobe oximetry under various conditions of exercise or high and low oxygen breathing is being advocated as a test of pulmonary function. Further investigations in this regard are needed. We have not used this and other "over all" function examinations as part of our screening because these tests evaluate the cardiorespiratory system in its entirety from the mouth to the peripheral capillaries. If the response is abnormal one does not know whether this is due to ineffective mass exchange of air, increased dead space or functional residual capacity, inefficient intrapulmonary distribution, impaired diffusion of oxygen across the alveolar capillary membrane, the existence of abnormal venous arterial shunts, defective transport of oxygenated blood, or incompletely "arterialized" blood in the ear lobe.

Dr Nemur mentioned his experiences with occlusion of one branch of the pulmonary artery. I have had an opportunity to see this examination performed by a group in Sweden. Striking roentgenographic and physiologic observations have been possible with this method. The complexity of machinery, however, and maneuvers required for simultaneous bronchospirometry, cardiac catheterization, and angiocardiology define this technique as a research procedure rather than a routine function test.

Dr Woodruff has assumed correctly that our surgical program has been modified from time to time by results of pulmonary function studies. Four years ago, in Denver, we presented a paper dealing with a number of patients with marginal function who were treated by extrapleural pneumonolysis. Our studies had suggested that this is a most function sparing procedure. Similarly staged thoracoplasty has been largely abandoned in favor of subtotal pleurectomy because it was shown that the latter procedure is more easily tolerated from a respiratory standpoint. In individual patients as well, the surgical program has been delayed or altered according to changes in pulmonary function. Occasionally, results of studies from the laboratory stayed our hands when we had been willing to operate on the basis of clinical observations. More often, results of function studies encouraged us to proceed with surgery in patients who clinically appeared to have insufficient pulmonary reserve.

We agree with Dr Woodruff that the measurement of the vital capacity, as such, is of little value in estimating pulmonary reserve. We continue to measure this volume for the calculation of the air velocity index. Lesser impairment of vital capacity than of maximal breathing capacity is indicative of an obstructive ventilatory defect. Also, the vital capacity must be known if the percentage of this volume delivered during various time intervals is to be calculated.

We should like to thank the discussants for their comments.

ARTERIALIZATION OF THE CORONARY SINUS IN OCCLUSIVE CORONARY ARTERY DISEASE

III CORONARY FLOW IN DOGS WITH AORTICOCORONARY SINUS ANASTOMOSIS OF SIX MONTHS' DURATION

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THIS is a continuation of previous studies^{1 2} undertaken to investigate the physiologic changes which occur in the coronary circulation following chronic arterialization of the coronary sinus for a period of six months

Pratt⁴ and Roberts and co workers⁵ first described the concept of revascularizing the heart in occlusive coronary artery disease, by perfusing the myocardium in retrograde fashion, through the coronary sinus Beck and his associates⁶ developed this procedure to its present two stage technique It utilizes the placement of a free vein graft between the aorta and the coronary sinus at a first stage, followed by the subsequent partial ligation of the coronary sinus to 2 to 3 mm one month later

Our experiments in dogs in which the coronary sinus had been arterialized for four to eight weeks leave no doubt that arterialization of the coronary sinus favorably modifies the mortality following the acute occlusion of the left circumflex coronary artery Moreover, in these dogs, we were able to demonstrate that (1) the myocardium was perfused in retrograde fashion, (2) the myocardium extracted oxygen from the blood flowing retrogradely, and (3) there was an increase in the intercoronary collateral flow

It was obviously essential to study dogs in which the coronary sinus had been arterialized for prolonged periods of time to determine whether the retro perfusion of the myocardium, and the increase in interarterial collateral flow persisted We are therefore reporting our findings in a group of animals in which the coronary sinus has been arterialized for a period of six months An additional group of animals in which the coronary sinus is being arterialized for a period of twelve months will be reported upon

METHOD

Mongrel dogs, whose weights varied between 12 and 18 kg, were first prepared, using the two stage operation developed by Beck At a first stage, through a left sided approach, an external jugular vein graft was placed between the aorta and the coronary sinus Three to six weeks later, using a right sided approach, the coronary sinus was partially ligated over a probe to 2 to 3 mm Six months following the second stage, physiologic data were obtained The animals were anesthetized with intravenous sodium pentobarbital, after which artificial respiration was maintained, using a Paeophore respirator*

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attached to an endotracheal tube with inflated rubber cuff to supply intermittent positive pressure. The left chest was entered through the fourth intercostal space and all pleural and pericardial adhesions were divided. The vein graft was dissected and examined for patency. Only dogs with patent grafts were used. An umbilical tape was passed around the graft to facilitate its occlusion at the appropriate moment. A needle tipped polythene catheter attached to a water manometer was inserted into the coronary sinus after which coronary sinus pressure and blood sample for determination of oxygen content were obtained (Fig. 1). The graft was occluded for a period of two minutes, after which the experiment was repeated (Fig. 2). The circumflex branch of the left coronary artery was dissected to its origin and ligated. An incision was made in the wall of the artery distal

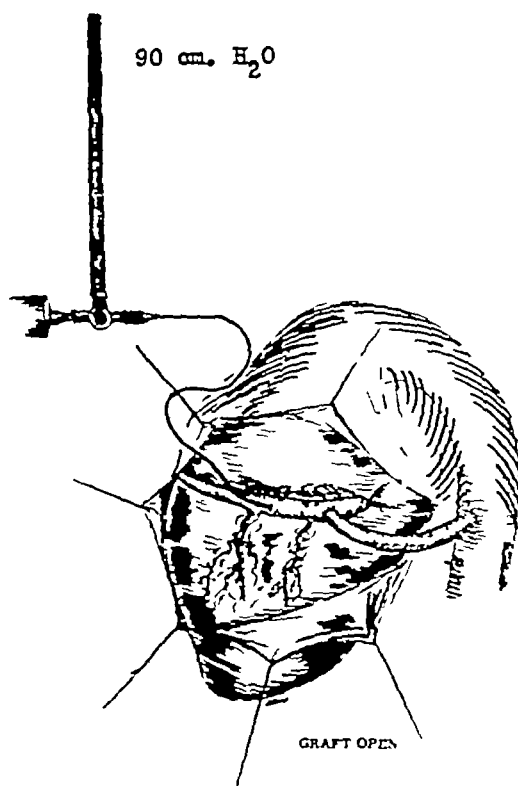


FIG. 1.—A needle tipped polythene catheter attached to a water manometer is inserted into the coronary sinus (indicated by the slit of partial ligation). Coronary sinus pressure is maintained at 100 mm. Hg. The recorded readings are corrected by adding the rise of the manometer above the level of the coronary sinus measuring this distance and totaling it with the manometric value.

to the ligature through which it was cannulated. A sample of retrograde circumflex artery or cranial flow was collected (Fig. 3) simultaneous samples being obtained from the circumflex posterior artery for determination of oxygen content and saturation data. The rate of retrograde blood flow was then recorded. At this point the graft was clamped for the experiment was repeated (Fig. 4). The distal circumflex artery was cannulated for flow for two minutes during which two minute retrograde flow was recorded prior to the collection of blood samples for oxygen content. This was repeated as in the previous experiments on the left to right flow and the rate of retrograde flow of oxygen content was gradually increased over a period of 10 minutes.

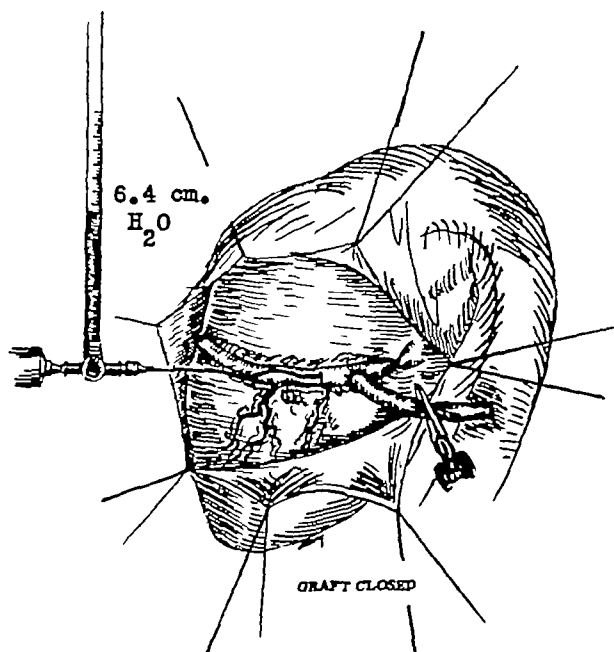


Fig. 2—A needle tipped polythene catheter was previously inserted into the coronary sinus proximal to the site of partial ligation and attached to a water manometer. The aorta to coronary sinus vein graft is occluded and the fall in coronary sinus pressure is recorded. Following occlusion of the graft a period of two minutes is allowed to elapse before a blood specimen is obtained for determination for blood oxygen content.

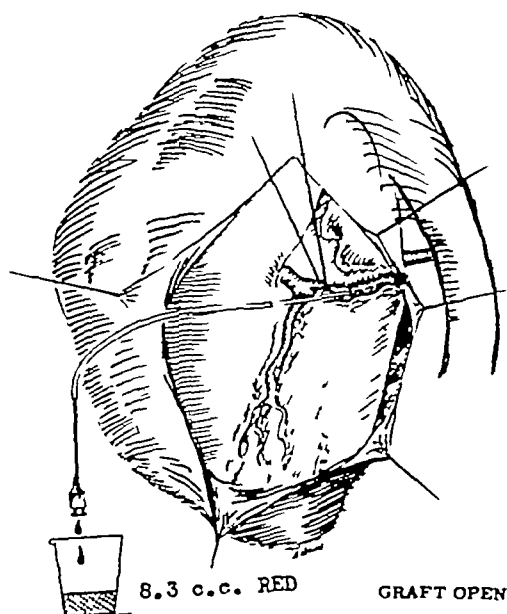


Fig. 3—The circumflex branch of the left coronary artery is carefully dissected to its origin where it is ligated. The artery is incised distal to the ligation and a polythene catheter is inserted. Retrograde blood flow from this distal segment is recorded and blood samples are secured for the determination of oxygen content.

RESULTS

A Retrograde Blood Flow From the Distal Cut End of the Ligated Circumflex Branch of the Left Coronary Artery—The results are shown in Table I. With the graft open, retrograde blood flow ranged from 30 to 180 c.c. per minute, with an average of 83 c.c. per minute.

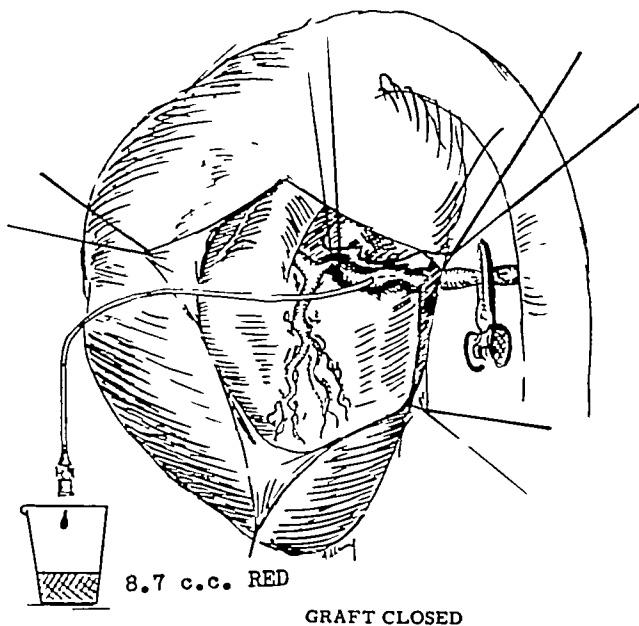


Fig. 4.—The left circumflex artery was previously ligated at its origin and the distal end was cannulated with a polythene catheter. The aorta to coronary sinus vein graft is occluded and the distal end of the transected coronary artery is allowed to bleed freely through the cannula for a period of two minutes during which time minute blood flow is recorded. At the conclusion of this interval of time a blood sample is secured for the determination of blood oxygen content.

Following the occlusion of the aorticocoronary sinus vein graft the retrograde circumflex artery blood flow remained unchanged in all dogs except Dog 6, in which it increased significantly. These values ranged from 30 to 170 c.c. per minute with an average of 87 c.c. per minute. Dog 6 will be discussed later, with respect to findings at post-mortem examination.

B Oxygen Content of the Retrograde Blood Collected From the Distal End of the Transected Circumflex Artery—The results are shown in Table I. The values for the oxygen content of this retrograde blood ranged from 10.0 to 17.4 volumes per cent with an average of 14.6 volumes per cent. Concomitant samples of aortic blood ranged from 16.4 to 19.1 volumes per cent with an average of 17.7 volumes per cent.

Upon careful inspection of these data it may be observed that the animals divide themselves into two groups. In Dogs 4, 5, and 6 the retrograde circumflex blood oxygen content closely approximates that of aortic blood and is therefore arterial in nature. In Dogs 1, 2, and 3 the retrograde circumflex blood oxygen content approximates that of the pulmonary artery, and therefore represents mixed venous blood. In none of these animals was the retro-

TABLE I RETROGRADE BLOOD FLOW AND OXYGEN CONTENT OF BLOOD FROM THE DISTAL CUT END OF THE LIGATED LEFT CIRCUMFLEX ARTERY IN SINUS ARTERIALIZED DOGS OF SIX MONTHS DURATION WITH THE GRAFT OPEN AND CLOSED

DOG NO	GRAFT OPEN CIRCUMFLEX ARTERY		GRAFT CLOSED CIRCUMFLEX ARTERY		AORTIC BLOOD			PUL. ARTERY
	FLOW CC/MIN	O. CONT VOL. %	FLOW CC/MIN	O. CONT VOL. %	CONT VOL. %	CAP VOL. %	SAT %	CONTENT VOL. %
	Approx							
1	18	10	17	14.9	16.9	16.5	100	11.6
2	9	13.2	9	18.3	16.9	16.9	100	12.4
3	4	14.8	3.8	15.1	19.1	19.3	99	14.8
4	7.5	17.2	7.5	17.0	19.0	19.9	95.5	15.5
5	3	15.3	3	17.2	16.4	17.8	92	13.5
6	8	17.4	12	17.2	17.8	17.3	100	12.3
Aver	8.3	14.6	8.7	16.6	17.7			13.4

grade blood oxygen content within the range of the normal coronary sinus blood as was invariably found in the dogs in which the coronary sinus had been arterialized for one to two months.

Following the clamping of the vein graft the blood oxygen content rose further toward that of aortic blood. These values ranged from 14.9 to 18.3 volumes per cent with an average of 16.6 volumes per cent. In Dogs 4, 5 and 6, these values remained practically unchanged. In Dogs 1, 2 and 3 there was a definite rise in blood oxygen content toward arterial levels.

C. Mean Coronary Pressure and Blood Oxygen Content—The results are shown in Table II. With the graft open the mean coronary sinus pressure ranged from 85 to 100 cm. of water, with an average of 90 cm. of water. The oxygen content of the arterIALIZED coronary sinus blood ranged from 14.9 to 19.6 volumes per cent with an average of 17.4 volumes per cent.

TABLE II MEAN CORONARY SINUS PRESSURE AND OXYGEN CONTENT OF CORONARY SINUS BLOOD IN SINUS ARTERIALIZED DOGS OF SIX MONTHS DURATION WITH THE GRAFT OPEN AND CLOSED

DOG NO	GRAFT OPEN CORONARY SINUS		GRAFT CLOSED CORONARY SINUS		PUL. ARTERY	AORTA CONTENT VOL. %
	PRESS CM. H ₂ O	O. CONT VOL. %	PRESS CM. H ₂ O	O. CONT VOL. %	CONTENT VOL. %	
1	85	16.7	6.0	4.8	11.6	16.9
2	90	16.6	6.5	6.0	12.4	16.9
3	90	18.8	6.5	9.8	14.8	19.1
4	85	19.6	6.0	8.9	15.5	19.0
5	90	14.9	6.0	10.3	13.5	16.4
6	100	17.9	7.5	9.7	12.3	17.8
Aver	90	17.4	6.1	8.3	13.4	17.7

Following the occlusion of the aorticocoronary sinus vein graft coronary sinus pressure rapidly fell to normal. The pressures ranged from 6.0 to 7.5 cm. of water with an average of 6.4 cm. of water. The coronary sinus blood oxygen content ranged from 4.8 to 10.3 volumes per cent with an average of 8.3 volumes per cent.

DISCUSSION

The most striking finding of the experiment revolves around the marked changes which occur in the coronary circulation between the second and sixth

month in animals in which the coronary sinus has been arterialized. In the coronary sinus arterIALIZED animals of four to eight weeks duration we were able to demonstrate unmistakable evidence of retroperfusion of the myocardial capillary bed. In the present series of coronary sinus arterIALIZED animals of six months duration we were unable to demonstrate this retroperfusion of the capillary bed. This finding was further verified by injection studies performed *in vivo*. In these experiments a colloidal suspension of carbon particles was injected into the aorticocoronary sinus vein graft of a few animals under arterial pressures of 120 to 150 mm. after the circumflex coronary artery had been ligated. Under these circumstances minimal injection of the left ventricle was noted whereas the rapid and abundant filling of the pericardial and endotheoracic vessels was striking.²

These injection studies suggested that arterIALIZATION of the coronary sinus produced an increase in the extracardiac collateral anastomosis. This increase in the coronary collateral circulation was further substantiated by our physiologic studies and was demonstrated in our series of both two- and six-month animals.

In our previous studies¹ we have been able to demonstrate the presence of only a small amount of intercoronary collateral flow in normal dogs. In those animals retrograde circumflex coronary arterial flow was small in quantity measuring approximately 17 cc. per minute and was arterial in nature. The arterial nature of this blood demonstrated its origin to be from neighboring coronary arteries and therefore represented a measure of the intercoronary arterial collateral flow.

In a series of animals in which the coronary sinus had been arterIALIZED for four to eight weeks these findings were significantly changed.² With the graft open the coronary sinus contained arterIALIZED blood under systemic pressures. Concomitantly retrograde circumflex arterial blood flow averaged 122 cc. per minute which represented a significant increase over the normal. Moreover this retrograde blood differed from that of the normal animal in that it was now markedly deoxygenated, the oxygen content being within the range of that of normal coronary sinus blood. The desaturation of this blood clearly demonstrated the retroperfusion of the myocardial capillary bed with the extraction of oxygen by the myocardium. Following the occlusion of the aorticocoronary sinus vein graft the retrograde circumflex coronary arterial blood flow decreased to 71 cc. per minute and again became highly oxygenated. The arterial nature of this blood again signifies that it has arisen from intercoronary anastomoses. This retrograde coronary arterial blood flow represents a fourfold increase above the expected normal retrograde flow values and probably represents a significant increase in the intercoronary arterial anastomotic flow.

In the present series of animals in which the coronary sinus has been arterIALIZED for six months the findings are significantly changed from those in the four to eight week group.

With the graft open the retrograde blood flow averages 83 cc. per minute. In both of these animals (Dogs 4, 5 and 6) this retrograde blood is highly oxygenated, the oxygen content of which is within the range of concomitantly

drawn samples of aortic blood. The oxygen content of the retrograde coronary arterial blood in these three dogs averaged 16.6 volumes per cent, as compared to an average oxygen content of 17.7 volumes per cent in concomitantly drawn samples of aortic blood. The arterial nature of this blood demonstrates that it has originated from neighboring unligated arteries. This then demonstrates an increase in intercoronary collateral flow. In the remaining three animals, the oxygen content of the retrograde blood averaged 13 volumes per cent, as compared to an average oxygen content of 17 volumes per cent in concomitantly drawn samples of aortic blood, and 13 volumes per cent in simultaneously drawn samples of pulmonary arterial blood. This retrograde circumflex arterial flow is therefore within the range of pulmonary arterial, or mixed venous blood. It in no way approximates the highly desaturated blood found in the two-month group. Rather, it is suggestive of an incomplete loss of retroperfusion, with mixing of blood from interarterial and capillary bed origin.

Following the occlusion of the aorticocoronary sinus vein graft, the retrograde circumflex arterial blood flow averages 8.7 c.c. per minute, which is insignificantly changed from that with the graft open. The oxygen content of the retrograde blood in Dogs 4, 5, and 6 remains unchanged and is persistently arterial in nature. The oxygen content of the retrograde blood in Dogs 1, 2, and 3 rises toward that of the aortic sample, much the same as was noted in our four- to eight-week animals.

Strikingly, then, in the animals in which the coronary sinus has been arterialized for six months the retrograde circumflex arterial flow is unchanged by the opening or occlusion of the aorticocoronary sinus vein graft. The oxygen content of this blood, too, is unaffected by the presence or absence of the vein graft in 50 per cent of the animals and is only minimally affected by the presence of a functioning graft in the other 50 per cent. In other words, no significant contribution to the retrograde circumflex arterial flow could be demonstrated to arise from the arterialized coronary sinus.

Thus, to all intents and purposes, the retrograde circumflex arterial blood flow averages 8.5 c.c. per minute and is arterial in nature. This retrograde flow represents a fivefold increase above the expected normal value and demonstrates a significant increase in the intercoronary arterial anastomotic flow. This finding is unaffected by the opening or the occluding of the vein graft. Moreover, a significant retroperfusion of the myocardial capillary bed cannot be demonstrated in this group of animals.

These findings serve to confirm those recently reported by Eckstein and his associates.¹⁰ These workers noted that the retroperfusion which existed in dogs in which the sinus had been arterialized for four weeks could no longer be demonstrated after three months. At the end of this period of time, they too found the retrograde circumflex coronary arterial blood to be arterial in nature and therefore postulated its origin to be from interarterial collaterals. Moreover, by injecting bromine gelatin into the circumflex artery they were able to demonstrate small intercoronary anastomoses. This finding has been demonstrated by Angulo¹¹ using the previously described colloidal suspension

on carbon technique for injection. Eekstein and co-workers also demonstrated that the blood flowing in retrograde fashion from the distal cut end of the ligated circumflex artery probably has its chief origin in the anterior descending artery. They were unable to demonstrate any significant contribution to this retrograde blood flow by the arterialized coronary sinus.

The mortality rate following circumflex coronary arterial ligation also increased significantly in the six-month group of animals as compared to that of the four- to eight-week group. For the purposes of this comparative mortality report those animals living for four hours and then sacrificed for injection studies are classified as survivals. In our normal control group of animals, ligation of the circumflex coronary artery at its point of origin from the main trunk resulted in a 90 per cent mortality. In our group of coronary sinus arterialized animals of four to eight weeks' duration this mortality rate fell to 20 per cent. In the present group of animals in which arterialization of the coronary sinus existed for six months, ligation of the circumflex coronary artery is attended by a 66 per cent mortality. This then demonstrates a progressive decrease in the protection to the heart afforded by the arterialized coronary sinus over the six-month period.

The retrograde circumflex arterial flow, with the graft open and closed, in Dog 6 bears comment. In this animal the retrograde flow increased from 8 to 12 c.c. per minute following occlusion of the graft. This was not just an isolated observation but was repeated and verified three times. Eekstein and associates¹⁰ also described this increase in retrograde flow following clamping of the graft in several dogs in which the sinus had been arterialized for longer than three months. At post mortem examination of this heart specimen we noted that the ostium of the coronary sinus had only been reduced to 4 mm. in contradistinction to a reduction of the coronary sinus ostium to 2 mm. in the remaining specimens. Possibly the 4 mm. ostium produced an arteriovenous fistula of such magnitude that occlusion of the graft by obviating the fistula improved the inflow of blood into the unligated anterior descending coronary vessel and thereby increased the interarterial collateral flow. It is interesting in which this increase in retrograde flow following occlusion of the graft was demonstrated the ostium of the coronary sinus was similarly ligated to only 4 mm.

The favorable effects produced by arterialization of the coronary sinus apparently regress over a period of six months. Further physiologic studies on animals in which the coronary sinus has been arterialized for a twelve-month period are now in progress and will be presented at a future date.

SUMMARY

1. Retrograde blood flow and oxygen content from the distal cut end of the ligated and transected circumflex artery have been determined in dogs in which the coronary sinus has been arterialized for a period of six months. These determinations have been performed with the graft open and closed.

2 With the graft open coronary sinus pressure is within the range of systemic pressure and the coronary sinus blood is of high oxygen content

3 When the graft is occluded coronary sinus pressure and blood oxygen content revert to normal levels

4 With the graft open, retrograde circumflex arterial blood flow is five times greater than the expected normal but is less than that present in the four to eight-week group of animals. This blood is arterial in nature, approximating the oxygen content of aortic blood. This retrograde blood flow is probably due to an increase in the interarterial coronary collateral circulation.

5 Occlusion of the graft does not significantly alter these findings which points toward its dispensability after six months.

6 The mortality rate following circumflex coronary arterial ligation, which was 20 per cent in the four to eight-week sinus arterialized animals rose to 66 per cent in the six-month group.

7 Retroperfusion of the myocardial capillary bed, which was demonstrated in the four to eight week animals could no longer be demonstrated in the six-month group of animals.

8 A significant fivefold increase in the retrograde blood flow occurring via intercoronary arterial anastomoses persists between the second and sixth month after arterialization of the coronary sinus.

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THE HEALING OF RAW LUNG SURFACES AFTER EXPERIMENTAL SEGMENTAL RESECTION

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SINCE first described by Churchill and Belsey¹ in 1939 and subsequently modified by Blades,² Clagett and Deterling,³ segmental pulmonary resection has received increasing attention. These pioneer efforts supplemented greatly by the important anatomic studies of Bowden and his associates^{4, 5} enabled Overholt and others to extend the technique and principles of segmental resection making virtually all pulmonary segments susceptible to such excision. The obvious advantages and desirability of preserving a maximum of normal lung parenchyma and thus greater function need no amplification. The initial and the chief indication for segmental resection was bronchiectasis, itself a segmental disease. Subsequently the indications for segmental resection have been expanded to include many other types of pulmonary pathology including tuberculosis.

In sharp contrast to total lobectomy and pneumonectomy the earlier reported series of segmental pulmonary resections demonstrated a uniformly higher incidence of complications in the form of fluid accumulation, pneumothorax, incomplete or delayed re-expansion, bronchopleural fistula and empyema.¹¹ Indeed Ochsner and his co-workers¹² at one time concluded that the frequent occurrence of such complications limited the usefulness of segmental resection. That these complications are more prone to develop when dealing with suppurative conditions is undoubtedly significant.^{11, 14} The use of the intersegmental vein as an anatomic guide to segmental resection demonstrated by Ramsey¹ and stressed by Chamberlain¹¹ and others⁸ and various methods to pleuralize the raw lung surface^{1, 16} have been suggested to overcome this high complication rate. More recent reports^{4, 16} indicate a gratifying reduction in complications and lead Chamberlain¹⁶ to conclude that the morbidity following segmental resection is no greater than in total lobectomy.

Since the raw lung surface that remains following segmental resection is not present after lobectomy unless an incomplete fissure must be transected it might seem that a causal relation exists between it and the complications which have been noted. This report is concerned with experimental studies in dogs to evaluate the immediate and ultimate fate of the raw lung surface, and attempt to assess various methods of treating such wounds.

Clinically Abbott¹⁷ opposed the use of fibrin foam or other coagulating materials, fearing that such agents might lead to clotting hemothorax. He did, however, advocate suturing the remaining segments to remaining intact lobes thereby preventing rotation of the preserved segments and subsequent

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bronchial obstruction. Most observers have been in agreement with Clagett and Deterling³ that the raw lung surface should not be closed by suture, feeling that such a technique predisposes to obstruction, possible bronchiectasis, and unnecessary loss of lung parenchyma. The use of pleural grafts or flaps to cover the raw surface as practiced by some has already been mentioned.

Experimental studies of lung wounds have been few, the first reported being those by Olch and Ballou¹⁸ in 1929. They made simple incised wounds in the lungs of dogs and closed the visceral pleura by suture. No adhesions between the parietal pleura and the scar on the visceral pleura were observed, and they concluded that a scar of lung tissue resembles a scar elsewhere in the body, save for the collapse of adjacent alveoli caught in the contracting and retracting scar. They also concluded from similar experiments, in which Trypan blue was given intravenously and intratracheally, that such scars arising from simple incision and suture have their origin from the supporting framework of the lung and that the cells lining the alveoli do not contribute to the scar. Daniel²⁰ investigated the effect of bullet wounds upon the lung, but, since he was concerned only with the immediate results of such trauma, no observations on their subsequent course or healing were made. Bailey, Ingraham, Swenson, Lowrey, and Beiring¹ created simple incised wounds of the lung in cats and monkeys, placing fibrin foam with thrombin in the wound and then suturing the visceral pleura. They felt that bleeding from such wounds was controlled by these agents and that tissue reaction to the hemostatic material was mild. No control series without the use of fibrin foam and thrombin was made; thus no comparative evaluation of immediate and ultimate tissue reaction was possible. Montgomery²² created wedge-like wounds in the lungs of cats and sutured the visceral pleura after the wedge of tissue had been removed. As small areas of denuded lung tissue remained between the pleural sutures, he made observations upon the restoration of the continuity of the pleura in these small areas. He concluded that the alveolar endothelium²³ was chiefly responsible for the formation of fibroblasts and of collagen. The generally accepted concept that lung tissue is incapable of regeneration was also challenged by this writer since it was thought that the terminal bronchioles did produce "buds" and regenerated from such areas of bronchial budding.²⁴ Joannides, Hesse, and Joannides²⁵ studied the effect of crush wounds, sutured wounds, and wedge resections, as well as the irritative effects of suture materials on the lungs of dogs. Valle and Mider²⁶ also investigated the reaction of lung to various suture materials in dogs. In addition they studied wounds created by clamping an edge of a lobe, resecting the lung distal to the clamp and closing the defect by a continuous stitch over the clamp which was then removed.

In all of the above experiments it is quite apparent that the wounds produced either by simple incision, wedge resection, or crushing were made without regard to segmental pulmonary anatomy. As a result division of bronchovascular structures occurred of necessity. While this in no way detracts from the observations concerning the healing of such wounds, it cannot be assumed that the raw lung surface created by segmental resection heals in a

similar manner. The only experimental study on the healing of the raw lung surface, created by segmental resection in cats, is that of Findlay²⁰ whose findings will be discussed later.

MATERIALS AND METHODS

Twenty-one dogs weighing between 12 and 20 kg were employed for these experiments. They were divided into two groups consisting of 8 dogs in Group I and 13 dogs in Group II.

Group I (8 dogs)—After anesthetization with intravenous Nembutal the right or left chest was shaved, an endotracheal tube inserted, and the dog suitably positioned for thoracotomy on an animal operating table. The skin was prepared with Kalmerid and draped with sterile towels and sheets. An aseptic technique was maintained throughout, and interrupted 4-0 black silk sutures were used. The thorax was entered through an intercostal incision, and upon opening the pleural cavity, the endotracheal tube was attached to a respirator delivering an intermittent flow of air under positive pressure, the latter being regulated by a water manometer. In each instance a raw lung surface was created by performing a segmental resection the superior segment of either the right or left diaphragmatic lobe being removed. Dissection was started with division of the pulmonary ligament up to the inferior pulmonary vein to facilitate the resection. The superior segmental vein of the diaphragmatic lobe was identified and cleared following which the superior segmental artery was freed up doubly ligated at its origin from the lobar artery and transected between the two ligatures. The superior bronchus was dissected out ligated with transfixion suture at its branching from the lobar bronchus clamped distally and transected between the ligature and clamp. After division of the artery and bronchus the previously cleared segmental vein was doubly ligated and transected between the two ligatures. In most instances it was possible to identify and preserve the intersegmental vein which was used as a guide to the intersegmental plane. With gentle traction applied to clamps placed on the transected bronchovascular structures, dissection of the intersegmental plane was begun at the hilum and with gentle blunt dissection the superior segment was removed the limiting visceral pleura being transected as the dissection proceeded peripherally. Following removal of the segment, the remaining raw surface was covered with a warm saline moistened gauze sponge and gentle pressure applied for two minutes after which the sponge was removed and the raw surface examined for an leak and bleeding. A soft rubber catheter was introduced into the pleural cavity through the thoracotomy wound and the chest wall was closed in layers around it after which the thorax was aspirated through the catheter which was then removed. The dogs were placed on the operative side following surgery and were left in this position until they recovered from the anesthesia. No chemotherapy or antibiotics were employed locally or systemically in any case. The size of the raw area created varied according to the size and weight of the dog; thus the triangular defects produced ranged from 9 to 15 square centimeters. In two instances re-exploration was carried out at the end of twenty-four hours (one day) in one dog and at twenty-two hours (three days) in another. At re-exploration observations

were made concerning the state of the previously created raw lung surface and resection of the remaining basilar segments was performed to obtain the specimens. These two dogs were subsequently utilized for contralateral segmental resections. Of the remaining 6 dogs, 4 had staged bilateral segmental resections and 2 expired, one during and one twelve hours after unilateral segmental resection. Thus in 8 dogs, 12 segmental resections were performed, and 2 dogs had additional lobectomies of the remaining basilar segments. The 6 remaining dogs were sacrificed at varying intervals and post mortem examination of the thorax was made immediately, noting the presence or absence of pneumothorax, effusion, adhesions or evidence of infection. The lungs were removed in toto and inflated to approximately normal size by the endobronchial infusion of a modified Karseling's solution, in which they were then placed for twenty-four hours, after which appropriate blocks were cut, placed in ten per cent formalin solution and prepared for microscopic study. Sections were made with hematoxylin and eosin, Mallory's trichrome, and Verhoeff elastic stains. In this manner gross and microscopic observations were available at 1, 3, 5, 10, 14, 21, 41, and 180 days postoperatively, and furnished information concerning the fate and healing of such wounds as well as furnishing a control for the experiments carried out in the second group of dogs.

Group II (13 dogs).—All dogs in this group were subjected to segmental resection in a manner identical to that described for the Group I animals. After removal of the superior segment, the remaining raw lung surface was treated by one of the following methods: Gelfoam without thrombin, Gelfoam with thrombin, Oxveel (cotton type), Oxveel (gauze type), and fascia obtained from the anterior rectus sheath. In two dogs attempts were made to cover the raw area with a free graft of parietal pleura or with visceral pleura swung from the surface of the resected superior segment. In the remaining animals a "fibrin gel" was employed to cover the wound surface. This was prepared by mixing 5.0 cc. of a 1.5 per cent solution of fibrinogen* with 1.0 cc. of thrombin† solution (10 units per cubic centimeter). This mixture resulted in a gelatinous clot within twenty seconds. It could not adequately be applied to the wound surface, however, since it would not adhere. After various trials it was found that the simultaneous application of these two agents in a fine spray by means of atomizers, so that the small droplets united on the wound surface, resulted in an adherent clot the thickness and size of which could be controlled. As in the previous group, most of these dogs underwent staged bilateral surgery, a total of 18 segmental resections being performed. No antibiotics were employed in this group and early observations on the effects of the material used to cover the wounds were not made. The dogs were sacrificed so that observations at 14 and 21 days were available. The specimens were examined in a manner similar to that outlined in Group I.

OBSERVATIONS

General.—At operation several observations were made. With deflation of the diaphragmatic lobe, re-inflation after occlusion of the superior segmental bronchus resulted not only in rapid inflation of the basilar segments, but also

*Fibrinogen (Bovine). The Armour Laboratories.
†Sterile Thrombin. The Upjohn Company.

in almost simultaneous inflation of the superior segment. This transpired with such rapidity that only a transient and fleeting plane of demarcation was noted between the basilar and superior segments. Ligation of the superior segmental artery uniformly caused striking blanching of the entire superior segment. In no instance was bleeding or any leak from the raw surface of any significance. After application of the moist sponge bleeding had either stopped or was manifest by only a scant slow oozing. Similarly the initial frothing caused by escaping air was either absent or nearly so.

Group I (Gross) At 1 day the wound presented a soft smooth dull red surface having a hemorrhagic fibrinous appearance. No pneumothorax or adhesions were noted. A small amount of serosanguineous fluid was present in the costophrenic recess. At 3 days adhesions and pneumothorax were still absent. The wound was smooth and glistening as though covered by a thin serosal layer which could be stripped off. The surface was firm to palpation and considerable clearing of the dark plane of resection to a lighter shade had occurred (Fig 1 A and B). At 5 days delicate hilar adhesions between the inferior surface of the cardiac lobe and the wound were first noted and at 14 days these adhesions had increased so that usually the entire surface of the intersegmental wound was covered by the inferior surface of the cardiac lobe.



Fig 1—A. Appearance of left lower lobe three days after resection of the superior segment. The hemorrhagic fibrinous surface of the segmental plane is at the upper right. B. Close-up view of the same specimen. The beaded appearance characteristic of the intersegmental plane is apparent. A shining glistening membrane covers the wound.

These surfaces could readily be separated, the wound then presenting a glistening aspect suggesting a covering of serosa with a white opalescent sheen beneath which the lung tissue appeared a normal pink color (Fig 2). At 21, 41, and 180 days little change could be noted in the observations made at 14 days. The adhesions between the wound and overlying cardiac lobe did not appear either more marked or denser (Fig 3). When separated the wounds showed no evidence of contraction and presented the appearance of almost normal lung. The surface for a somewhat thicker milky translucent surface over the underlying parenchyma of the wound which also extended to involve the adjacent visceral pleura but to a lesser degree. In only one instance was an adhesion noted

between the wound and parietal pleura, and this was represented by a single, fine strand of tissue. One death resulted during operation from cardiac arrest, the other was associated with a tension pneumothorax, autopsy revealing disruption of the bronchial stump.

(Microscopic) At 1 day—Sections exhibited a predominantly exudative and vascular reaction. The wound surface was coated with a fibinous layer containing only occasional trapped red cells and leukocytes. Beneath this fibrin surface a densely cellular area involving the parenchyma and consisting of a very intense hyperemia was noted. Under low magnification this area appeared hemorrhagic.

Upon closer inspection at higher magnifications, however, it was evident that while some extravascular bleeding and hemorrhage into alveolar spaces had indeed occurred the chief response was one of massive engorgement of the alveolar capillaries to such a degree that many air spaces were themselves obliterated. Neutrophils and lymphocytes were present, but there was no significant leukocytic response. In the areas of marked hyperemia, the alveolar (or capillary endothelial) cells were enlarged with a denser, larger nucleus than is usually observed. No necrotic areas were seen and the neighboring bronchi were free of secretions and exudate although some red cells were present intraluminally (Fig. 5).

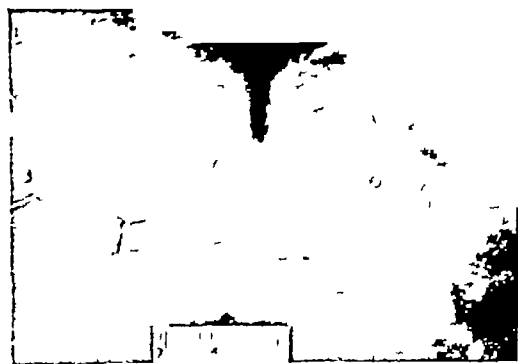


Fig. 2

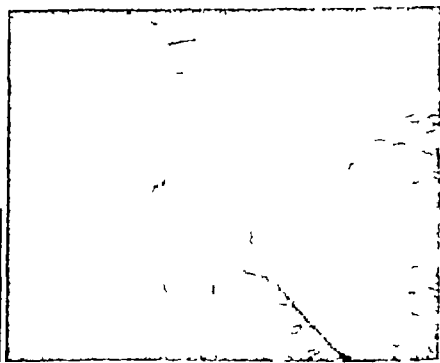


Fig. 3

Fig. 2—Appearance at 14 days. Cardiac lobe to the left, basilar segments on the right. The wound is pleuralized and hilar adhesions have formed.

Fig. 3—Appearance at 21 days. Only a fine scar separates the basilar segments and cardiac lobe. Such symphyses were frequent by 14 days.

At 3 days—The wound surface was composed of a laminated layer of fibrin between which small numbers of red cells and occasional leukocytes were noted. The underlying parenchyma no longer demonstrated the intense capillary hyperemia seen at 24 hours, and the alveoli though compressed contained air.

At 5 days—Much of the previously noted fibrin surface was absent, being replaced and invaded by large numbers of fibroblasts among which capillaries and small vessels were visible. The alveoli subjacent to this layer of fibroblastic proliferation still showed evidence of some compression and increased width of the alveolar walls. Within the alveoli were scattered red cells, occasional leukocytes, and numerous large histiocytes. Nearby bronchi contained moderate amounts of exudate and secretions (Fig. 6).

At 14 days—The wound surface was composed exclusively of fibroblasts and areolar connective tissue with an increased vascularity and being of somewhat greater thickness than previously. Alveolar lining cells abutting upon this

Fig. 1



Fig. 2

Fig. 2. Section through normal dog lung for comparison and to demonstrate pleura and subpleural layer. The pleural quilt is thin and not very vascular. Undulations are caused by subpleural connective tissue. Lymphatics are present in the lymphatics. (Masson stain, $\times 85$)

Fig. 3. Section of lung at 24 hours. Hemorrhage and marked alveolar hyperemia is present. Alveolar lining cells are present. Bronchi are clear of secretions. A fibrin layer coats the wound surface. (H. & E. stain, $\times 16$)

fibrinous layer. The alveoli are rounded or spherical in shape. Elsewhere, the alveolar walls are of normal thickness and the air spaces fully expanded. No alveolar or bronchial secretions or exudate were present (Fig. 7).

At 21 days. The wound presented an appearance similar to that described at 14 days, but there were fewer fibroblasts and a tendency toward stratification

into a superficial zone of areolar connective tissue, and a deeper zone of more densely arranged collagenous fibers. The superficial areolar layer extended to cover the visceral pleura surrounding the wound area, and the inner collagenous

FIG. 6



FIG. 7

Fig. 6.—Wound at 5 days. Fibrin coat still present and marked fibroblastic proliferation with capillary formation demonstrable. Alveoli are air containing, but still compressed with thickened walls. (Hematoxylin and eosin, $\times 200$.)

Fig. 7.—Wound at 14 days. Fibroblasts and vascularization more marked. Parenchyma is now essentially normal. (Hematoxylin and eosin, $\times 200$.)

Liver appeared continuous with the fibrous liver of the adjacent pleura. Appropriate stains failed to demonstrate any elastic elements in the wound, the normal elastic liver of the visceral pleura being deficient throughout the wound. The subjacent parenchyma showed no changes (Figs. 8 and 9).

At 180 days—The stratification noted at 21 days was more striking and there was a noticeable decrease in the vascularity and thickness of the wound surface. No elastic layer could be demonstrated. No other changes were present (Fig. 10).

Fig. 8



Fig. 9

Fig. 9. At 14 days, stratification of connective tissue occurred and fibrous tissue is more prominent. Elasticity is lost in the interstitial tissue. Normal alveoli are still present at the 1 ft. It is noted that the surface of the wound is far and deeper more densely arranged than the normal alveoli. Superficial are far and deeper more densely arranged than the normal alveoli. The vascularity of the wound (A) is less (Fig. 9).

Fig. 10. At 180 days, observations in this group were made only at 14 and 21 days. The gross findings were essentially as in the control group for this period. There is no difference between the intersegmental wound and the parietal pleura.

were invariably present except in the dogs treated with "fibrin-gel." The wound surfaces of all but the "fibrin-gel" treated dogs contained thicker, denser adhesions, and a tendency toward contracture of the wound surface was noted (Fig 11). Animals treated with the "fibrin-gel" spray showed little change

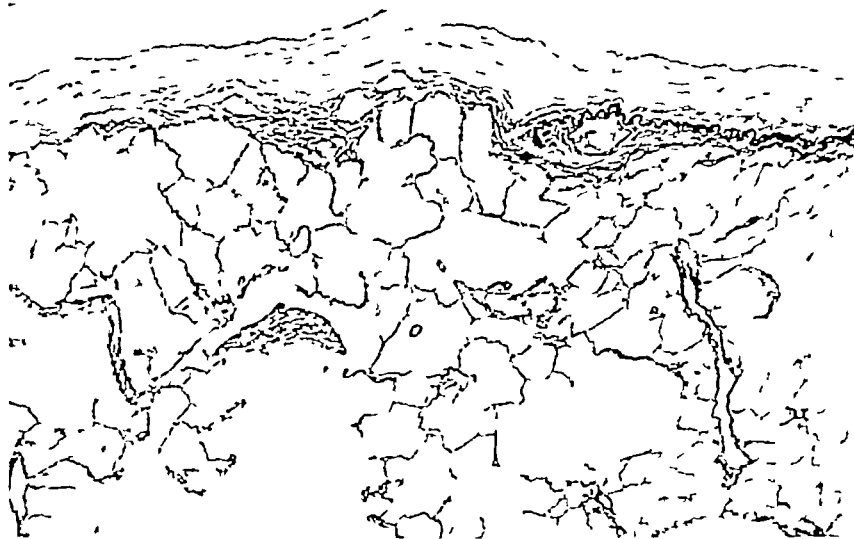


Fig 10—Wound at 108 days. Elastic layer of intact pleura is visible on the right. It is absent in wound surface on the left. The stratification is well demonstrated and the superficial areolar layer continues across normal pleura. (Verhoeff $\times 105$)



Fig 11—Wound at 14 days treated with Gelfoam. Reaction more marked than in control. Compare with Figs 2 and 3.

from the control group. Two deaths occurred in Group II animals. One dog treated with Gelfoam died nine days postoperatively of a massive empyema. The other dog, in whom a pleural graft had been attempted, expired on the fifth postoperative day of bilateral multilobar bronchopneumonia and focal atelectasis. Neither dog is included in the description.

(Microscopic) Sections of wounds treated by Gelfoam, Oxoid, and fascial grafts when examined at 14 days were similar to those of the control group.

after the same interval but the fibroblastic reaction was of increased intensity and the subjacent alveoli contained some exudate and were more vascular than present in the control group for this period. The fibrin-gel treated wounds appeared almost identical with the control group (Figs 12 through 16)

Fig. 11.

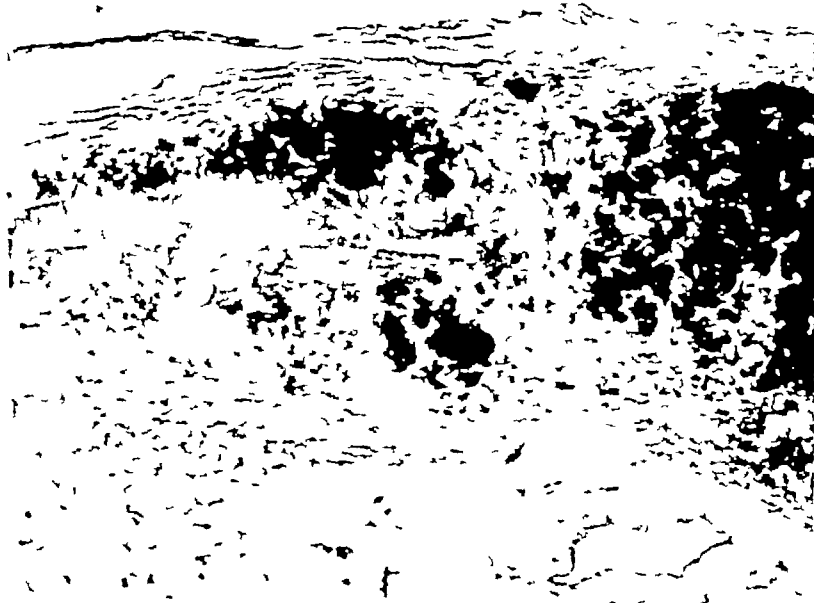
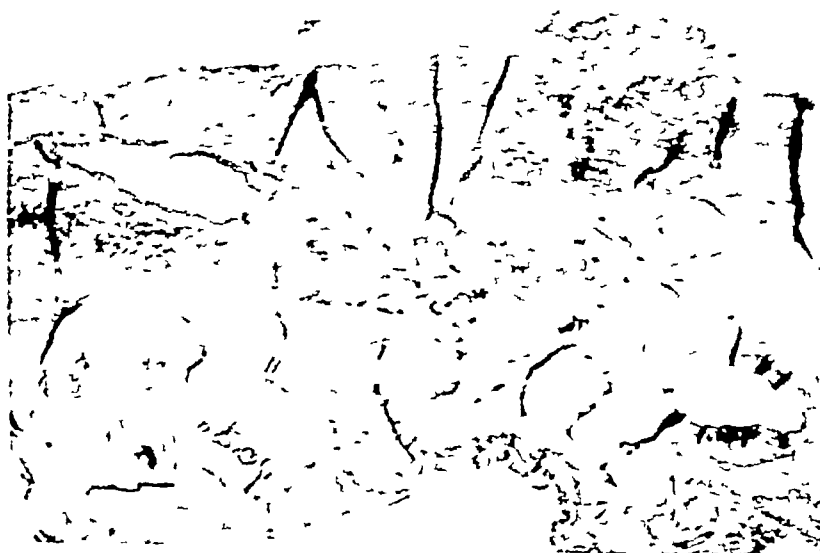


Fig. 12.

Fig. 13. (a) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (b) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (c) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (d) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (e) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (f) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (g) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (h) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (i) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (j) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (k) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (l) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (m) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (n) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (o) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (p) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (q) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (r) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (s) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (t) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (u) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (v) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (w) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (x) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (y) 14 days after resection. The reaction is characterized by a dense cellular infiltrate. (z) 14 days after resection. The reaction is characterized by a dense cellular infiltrate.

Fig. 14



Fig. 15

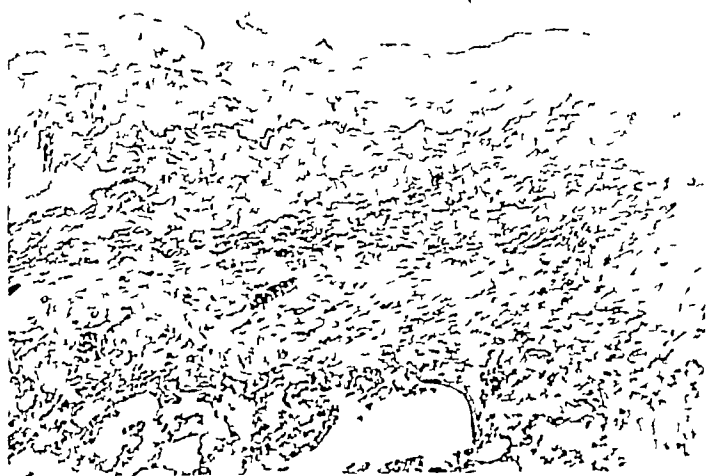


Fig. 16



Fig. 14—Sections through wound treated with fascia 14 days previously. The hyalinized fascia is apparent. The underlying alveoli are quite compressed. (Hematoxylin and eosin $\times 10$.)

Fig. 15—Section of wound treated with fibrin 14 days previously. There is slightly more reaction than in control but considerably less than with other agents employed. (Verhoeff $\times 10$.)

Fig. 16—Section through wound treated with fibrin 14 days previously, seen on the left. The normal alveolar pattern on the right contains an elastic layer deficient through the wound. (Verhoeff $\times 10$.)

DISCUSSION

Failure to obtain collapse of a segment when its bronchus was occluded is an example of active collateral respiration which is well developed and readily observed in dogs.²⁰ Segmental blanching immediately apparent after interruption of a segmental pulmonary artery is a physiologic demonstration of the end nature of these arteries and is further confirmation of anatomical descriptions that such vessels do not generally cross intersegmental planes.⁴



FIG. 17

FIG. 18

Fig. 17—Photograph at operation taken just after removal of superior segment. Pubbles of air can be seen.

Fig. 18—Same specimen as in Fig. 17 after application of fibrin gel which is quite thick near the hilum.

The absence of significant air leak or bleeding following segmental resections performed in these experiments made impossible any evaluation of the various methods used to treat the wounds thus produced with regard to these two factors. Attempts to pluralize by grafting the defects created met with technical failure since the thin pleura of dogs made it impossible to obtain an adequate sheet of intact membrane sufficient to cover the wound.

The only conclusion made regarding the other methods utilized to treat the raw lung surface is that the healing process in terms of adhesions, parenchymal reaction and fibrosis of the wound was more marked than in untreated control wounds at comparable periods of observation. The one exception was in those wounds treated with fibrin gel in which no significant difference was noted. Employment of fibrinogen and thrombin solutions to form a fibrin layer on these wounds was predicated upon the observation that fibrin is normally elaborated on such wound surfaces quite early, and that additional exogenous fibrin might be of value by acting as a seal to lessen bleeding and air leak. Since (1) after resections could not be evaluated, no conclusions concerning the efficacy of fibrin gel can be made in this respect. However, in the absence of significant reactions, further evaluation and refinement of this technique might prove fruitful (Figs. 17 and 18).

Observations upon the healing and fate of the untreated raw lung surface created by segmental resection indicate that the healing process is a function of the underlying parenchyma of the lung. The early alveolar hyperemia and exudation of fibrin on the wound surface, and later fibroblastic proliferation with capillary formation, must of necessity be dependent upon the parenchyma beneath the wound. Only when the wound possesses a well developed vascularity (14 days) do adjacent alveoli no longer reveal changes. At 21 days, healing is essentially complete, there being but moderate further maturation and condensation of the connective tissue between that time and six months. The "scar" at six months is essentially a reconstituted pleura, differing from the normal structure only in a somewhat thicker layer of connective tissue and the absence of an elastic layer.

Our studies thus tend to confirm those of Findlay²⁵ on the healing of such raw wounds, with two possible exceptions. First, it is felt that the early vascular reaction is greater than noted by this worker, although his earliest observations were made one day later than in those reported here. Second, although special studies to demonstrate mesothelial growth over the healing wound were not undertaken by us, the striking presence of the thin, glistening membrane covering these wounds at 3 days, suggests that such mesothelial surfaces cover the wound sooner than the 6 weeks as observed by Findlay.

The superficial layer of alveolar connective tissue which covers the wound as well as adjacent visceral pleura is quite similar to the thin epipleural "peel" frequently observed following therapeutic pneumothorax. Possibly, this reaction may result from manipulation and exposure of the lobe at the time of operation and need not be concerned with, or a function of, the healing wound per se.

We concur with the opinion of Valle and Mider²⁴ that regeneration of lung tissue as interpreted by Montgomery²² probably does not occur, since "bronchial budding" was not observed in any of the wounds studied by us. The observations of Watts and McDonald²⁶ on alveolar lining cells in bronchiectasis afford an explanation for the "bronchial budding" noted by Montgomery. It is felt that the marked fibrosis resulting from the type of wound produced by Montgomery, caused the formation of clusters of such alveolar cells which were mistakenly construed as evidence for bronchial and alveolar regeneration.

In discussing the subject of wound healing Harvey¹⁹ stresses that the phenomenon proceeds at a given velocity and cannot be expedited but that many factors may retard complete or stop the process. It is believed that the differences noted between the treated and untreated raw lung wounds studied by us support this fundamental concept. The larger depressed scars observed by others^{15, 22, 23, 24} when pulmonary wounds are made without respect to bronchovascular structures are also confirmatory evidence.

Segmental pulmonary resection creates a relatively vascular wound, does not transect bronchioles in the absence of infection or other complicating factors, the wound heals ideally with little reaction, without significant parenchymal destruction and results in a slightly modified pleura which is in fact the "scar" of such wounds.

SUMMARY

- 1 The significance of the raw lung surface created by segmental pulmonary resection is discussed
- 2 Previous reports concerned with the healing of experimentally produced pulmonary wounds are reviewed
- 3 Observations on the healing and fate of treated and untreated raw intersegmental lung wounds produced in dogs are presented
- 4 The special problems arising from wounds resulting from segmental pulmonary resection are discussed and compared with the healing of other types of pulmonary wounds

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BENIGN MESOTHELIAL CYST OF THE DIAPHRAGM

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PRIMARY tumors of the diaphragm are exceedingly rare, cystic tumors of the diaphragm are even less common, and benign mesothelial cysts of the diaphragm are medical curiosities. The most common mesothelial cyst in the chest, while not of diaphragmatic origin is the so called pericardial coelomic cyst or cardiophrenic angle cyst.

Granché¹ reported the first primary tumor of the diaphragm in 1868 as an incidental autopsy finding. In reviewing the literature in 1950, Samson and Childress² recorded 35 cases of primary tumors of the diaphragm, about half of these were malignant. A very small proportion of these cases were primary cysts and, as of 1949 (according to Aufses and Oseasohn³), there were only six cases of primary cysts of the diaphragm in the literature. Four of these cysts were lined by mesothelium and were considered congenital anomalies. Another one was a fibrous cyst lined by granulation tissue. This was considered tuberculous in origin and was successfully removed (Robson and Collins⁴). In the sixth case Scott and Morton⁵ reported the successful removal of a large cyst filled with chocolate-like fluid from the dome of the left diaphragm.

In 1949 Taggett and Johnson⁶ added to the literature a case in which the tumor was found in the dome of the right diaphragm and histologically was a "fibrous walled cyst containing putty like material."

To these seven cases Cruickshank and Cruickshank,⁴ in 1951, added two cases of mesothelial cysts. Both cysts were intradiaphragmatic and the authors were unable to find any similar cases in the literature.

Recently we encountered a case of mesothelial cyst of the diaphragm very much like those reported by Cruickshank and Cruickshank.

CASE REPORT

A 49 year old white man was first admitted to the Robert Packer Hospital on March 21, 1953. For almost three years he had suffered from intermittent chest pain which began in the back and radiated anteriorly into the hypochondrium. It has been present constantly for the month preceding admission and was aggravated by motion and deep breathing. At times it was necessary for him to sleep in a chair.

Other than this, his past history was unremarkable and the physical examination as well as routine laboratory studies disclosed no abnormalities.

A chest roentgenogram (Fig. 1), however, revealed a smoothly margined, homogeneous, globular shadow in the left leaf of the diaphragm anteriorly, containing a small plaque of calcium. Fluoroscopic studies showed the shadow to move with the diaphragm without alteration in size or shape. Barium studies of the upper and lower gastrointestinal tract failed to reveal any connections to the mass. Pneumoperitoneum was performed using 600 cc of air; this demonstrated conclusively that the mass was intimately attached to the diaphragm (Fig. 2).

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Fig 1—Chest film showing the smoothly margined homogeneous shadow in the left leaf of the diaphragm.



Fig 2—Chest film showing the smoothly margined homogeneous shadow in the left leaf of the diaphragm.

A thoriotomy was then performed with the patient in the left lateral position, using cyclopropine ether endotracheal anesthesia. The eighth rib as well as short segments of the seventh and ninth ribs were resected and the chest wound widely spread. In the dome of the diaphragm was a tense cystic structure 6 cm. in diameter located within and projecting above the substance of the diaphragm and extending medially to the esophageal hiatus. The diaphragm moved normally. An incision was made adjacent to the apparent limits of the tumor. The tumor, together with the attached diaphragm, was resected and the defect created in the diaphragm was repaired with a fascia lata graft. The phrenic nerve was crushed to immobilize the diaphragm temporarily. A catheter was placed in the costophrenic angle, brought out through a stab wound in the midaxillary line, and attached to an underwater seal. The wound was closed in layers with interrupted silk.



Fig. 3—Photomicrograph showing the delicate mesothelial lining of the cyst.

The postoperative course was entirely uneventful and the patient was discharged ten days after surgery. On follow up visits he had no complaints, seeming completely relieved of his chest pain.

Pathologically the tumor was a thin walled cystic structure, located within the substance of the diaphragm with muscle fibers spread out both above and below the cyst. The contents were slightly milky and histologically the lining was composed of well preserved mesothelium with only a few fibroblasts for support (Fig. 3). Diagnosis was benign mesothelial cyst of the diaphragm.

DISCUSSION

With the ever increasing utilization of routine chest x-rays it is expected that more tumors of the diaphragm will be disclosed. Since the majority of these tumors are malignant it is important that they be resected surgically without undue delay.

The diagnosis of these lesions may be difficult and can be made with certitude only by biopsy. X-ray alone is not sufficient for the establishment of a diagnosis. There are many aids in diagnosis including fluoroscopy, Lipiodol studies of the bronchial tree, barium studies of the upper and lower gastrointestinal tract, pycelograms and pneumoperitoneum. Of these the latter is one of the most helpful and its wider use has been suggested by Binney.²

It is unusual for mesothelial cysts to produce symptoms but at least one such case has been reported.

PATHOGENESIS

Mesothelial cysts of the chest are considered congenital in origin. The mechanism of their development has long been a subject of conjecture. Many theories have been advanced. Cruickshank and Cruickshank have "collected and systemized" the various embryological theories.

In their review of the six major theories they note that all these mesothelial cysts seem to arise from vagaries in the development of the mesothelial surfaces lining the primitive coelomic cavity and its three daughter cavities, the pericardial, pleural and peritoneal. From these findings they concluded a more appropriate nomenclature would be based primarily on the histologic type of cyst (mesothelial) and secondarily, on the anatomical site of origin (pericardium, pleura, peritoneum, cardiophrenic angle, diaphragm, etc.).

Further, they believe that cysts arising by entirely different embryologic processes from entirely different embryological structures should be classified in a similar manner and not by cyst contents. For example, the term serous cyst is indefinite enough to include bronchogenic, mesothelial and lymphangiomatous cysts.

Specifically, the diaphragmatic mesothelial cysts are thought to develop during the final closure of the pleuroperitoneal or pleuropericardial openings when mesothelial surfaces are approximated. A loculus may be formed by these two different mesothelial surfaces initiating a cyst or invagination of the free edge of an advancing mesothelial membrane may occur to form an intradiaphragmatic cyst of the type we are reporting.

SUMMARY

We have reported a case of benign mesothelial cyst of the diaphragm which was successfully removed. It appears to be the tenth primary cystic tumor of the diaphragm in the literature, the third intradiaphragmatic mesothelial cyst to be reported, and the only known intradiaphragmatic mesothelial cyst to produce symptoms which were completely relieved following excision of the tumor.

The embryogenesis of these cysts has been reviewed as well as the available diagnostic techniques.

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 Erratum

In the January issue of the JOURNAL, in the article by Dr Denton A Cooky and Dr Michael F DeBakey entitled Resection of the Thoracic Aorta With Replacement by Homo-graft for Aneurysms and Constrictive Lesions, pages 66 104, Tables IA and IB have been transposed. The table on page 90 enumerating Cases 1 7, should appear under the heading for Table IA on page 70. The table enumerating Cases 8 12 should be placed under the heading for Table IB on page 90.

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EXPERIENCES WITH EXTENDING THE INDICATIONS FOR THE USE OF TRACHEAL AND BRONCHIAL GRAFTS

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AN EXTENSIVE literature has already accrued relative to reconstructive surgery of the tracheobronchial tree. Daniel¹ initiated interest and enthusiasm in this field with his presentation before this society during the St. Louis meeting in 1947. He repeated and confirmed the initial experiments done by Taffel² in 1940, utilizing free grafts of fascia to cover windows created through the full thickness of the wall of the trachea. He then extended his investigation to include a study of the effect of spanning large gaps in the continuity of the tracheobronchial tree with various types of prostheses. Subsequent experience³ with applications of this type of procedure showed an almost constant tendency of the human trachea to become stenosed severely upon removal of the prosthesis. At the 1949 New Orleans meeting, Gubner⁴ described four highly successful instances of reconstruction of tuberculous bronchial stenosis with full thickness dermal bronchial grafts. In October, 1949, Robb and Bateman⁵ described the use of tantalum gauze covered with fascia to effect a repair of the cervical trachea. A successful repair of a traumatic stricture of the bronchus by excision and end-to-end anastomosis is reported by Griffith⁶ in 1949. The application of fascioplaryngeal procedures with end to end anastomosis of the trachea or bronchus was described in 1950 by Marsel and Dingwall⁷ and by Ferguson and Wild.¹⁰ In 1951, Mathew and Onstut¹¹ reported successful end to end anastomosis in the human after accidental division of the stem bronchus. Also in 1951, Pridson⁸ described the use of a dermal graft to correct a stricture of the trachea resultant to severe chest trauma. Methods of tracheal wall reconstruction were reported by one of us (O.A.A.) at the 1948 Quebec meeting of this Association. Quite large defects in the tracheobronchial wall were repaired following total removal of the right lung for extensive right upper lobe carcinoma with concomitant resection of the carina, tracheal wall and

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contralateral bronchial wall. With smaller defects, a transverse closure of the longitudinal ovoid defect constituted a satisfactory reconstruction without decrease in the lumen of the airway. In one patient, a very extensive defect was spanned by use of a prosthesis over which was placed a large pedicle pleural graft. In five instances, operations were performed for extensive carcinoma and, in one instance, for a bronchial adenoma which had extended across the carina down into the contralateral bronchus. This latter patient, who was reconstructed by suture without prosthesis or graft, lived almost four years following surgery free of any pulmonary symptomatology. She succumbed to an extensive neoplastic process in the liver, the exact nature of which could not be determined because of refusal of permission for post-mortem examination. The five patients with advanced carcinoma lived from two months to one year, all succumbing to either local or distal recurrence of the process. The repair of the tracheobronchial ovoid defect by transverse closure necessarily limited the amount of resection of tracheobronchial tissue adjacent to the neoplastic process. In addition, we experienced major difficulty during the postoperative period with the tendency for the airway to become partially or totally obstructed with any shift of the mediastinum. The patient in whom a glass prosthesis was placed because of the need to make a very extensive defect is being represented in this publication and will be discussed further in detail.

The initial use of dermal grafts by Gebauer was confined to the treatment of tracheobronchial lesions of the tuberculous type. In 1950, he¹⁴ wrote, "perhaps in time dermal grafts will be used more frequently for non-tuberculous tracheobronchial lesions than for the tuberculous type." This prophecy has been partially fulfilled with the reports of the use of grafts in the management of the effects of both acute and posttraumatic stricture. Occasional instances of the use of the procedure for the treatment of intra-bronchial tumors have been described in 1947 by Goldman¹⁵ and in 1948 by Langston and Fox.¹⁶ We wish to report our experiences with the utilization of grafting procedures in the management of nontuberculous lesions.

At the present time we feel that there are several indications for considering the application of grafts upon the trachea or bronchus. We submit an outline of these indications for consideration in Table I. There are 3 basic categories: (1) acute trauma, (2) various types of bronchostenosis, both tuberculous and nontuberculous, (3) in the management of various types of different neoplastic processes. There may later be added (4) congenital defects, such as partial agenesis of cartilage. Gebauer¹⁴ and Paulson¹² have emphasized that in some instances strictures need not be associated with suppurative destruction of the pulmonary tissue distal to the stricture. In such instances a repair of the stricture constitutes the only reasonable physiologic surgical approach. Such a situation occurred in Paulson's management of a posttraumatic stricture in which the injury was produced by severe external compressive force upon the thorax. We are reporting herein a similar situation resultant to the intrabronchial trauma incident to the long standing presence of a metallic foreign body; we assume

TABLE I. THE PRESENT INDICATIONS FOR CONSIDERATION OF THE POSSIBLE NEED FOR GRAFT-TYPE TRACHEAL CRAFT

1. Auto trauma
 2. Bronchogenic cysts with functional distal lung
 - A. Tuberculous structure
 - B. Nontuberculous structure
 1. Traumatic
 2. Infectious

{

Extrabronchial
Endobronchial
3. Tumors
 - A. Bronchogenic cysts
 - B. Adenoma
 - C. Carcinoma
 1. hilar with respiratory insufficiency
 2. Tracheal or carinal lesions
 3. Plait upper lobe lesions
4. Congenital partial agenesis?

but have no record of an authenticated instance of a similar situation which could be produced by a nontuberculous localized endobronchial infection. The third category, namely that of neoplastic processes, includes several subsidiary divisions. Any type of neoplasm involving the trachea itself allows one but little choice except to utilize some type of graft or graft plus prostheses. In fact it was a tracheal tumor which had been observed to exist for over two years which incited Daniel's initial interest in this problem. Both primary carcinomas and adenomas of the trachea and bronchial wall have been managed in this manner. We have had opportunity to utilize this approach in the management of a large bronchogenic cyst arising from the posterior tracheal wall. It has also been used in order to resect a more adequate margin of bronchial or tracheal tissue adjacent to neoplasms. Grafts have been used to reconstruct major defects produced by resection of the entire tracheal wall and contralateral bronchial wall produced by radical right total pneumonectomy for carcinomas arising in the region of the right upper lobe orifice. Finally we have found a very definite usefulness of the bronchial graft principle in the management of carcinomas arising in the hilum of a pulmonary lobe in patients whose respiratory capacity is insufficient to allow any more extensive resectional procedure than lobectomy.

TECHNICAL INDICATIONS

At the present time we have had opportunity to employ grafting procedures in 10 nontuberculous patients. The basic data relative to these 10 patients are summarized in Tables II A and III.

Case 1. Graft in Childhood.—In June 1951 an 11-year-old boy, W. W., presented himself for evaluation of his "bronchial asthma." The patient's chest had been free of any physical signs of disease until about 1 year prior to his admission to the hospital. For the ensuing 12 months he had had to have progressive symptoms of recurrent attacks of cough and wheezing. The right lung aerated very poorly on examination. On fluoroscopic and roentgenographic examination the right lung was seen to be compressed by a large mass of the entire right lung. The mass was seen to be of the same density as the normal lung. Bronchoscope

TABLE IIA SUMMARY OF CLINICAL EXPERIENCES WITH THE USE OF DERMAL GRAFTS IN OPERATIONS NOT INCORPORATING RADICAL RIGHT TOTAL PNEUMONECTOMY

	AGE	DIAGNOSIS	PROCEDURE	RESULTS
L F	4	Bronchogenic tracheal cyst	Local tracheal resection and dermal graft	Asymptomatic 16 months
W W	11	Benign stricture right main stem bronchus	Excision and plastic reconstruction with dermal graft	Asymptomatic 32 months
S H	52	Adenoma of carina and left main stem bronchus	Local resection of carina and bronchus with graft	Asymptomatic 7 months
J C	48	Extensive right upper lobe carcinoma with severe emphysema	Resection right upper lobe and adjacent tracheal wall with graft Post operative radiation therapy	No recurrence 9 months
P H	55	Left lower lobe carcinoma with pulmonary fibrosis and emphysema	Resection left lower lobe and medial portion left main stem bronchus with graft	No recurrence 5 months

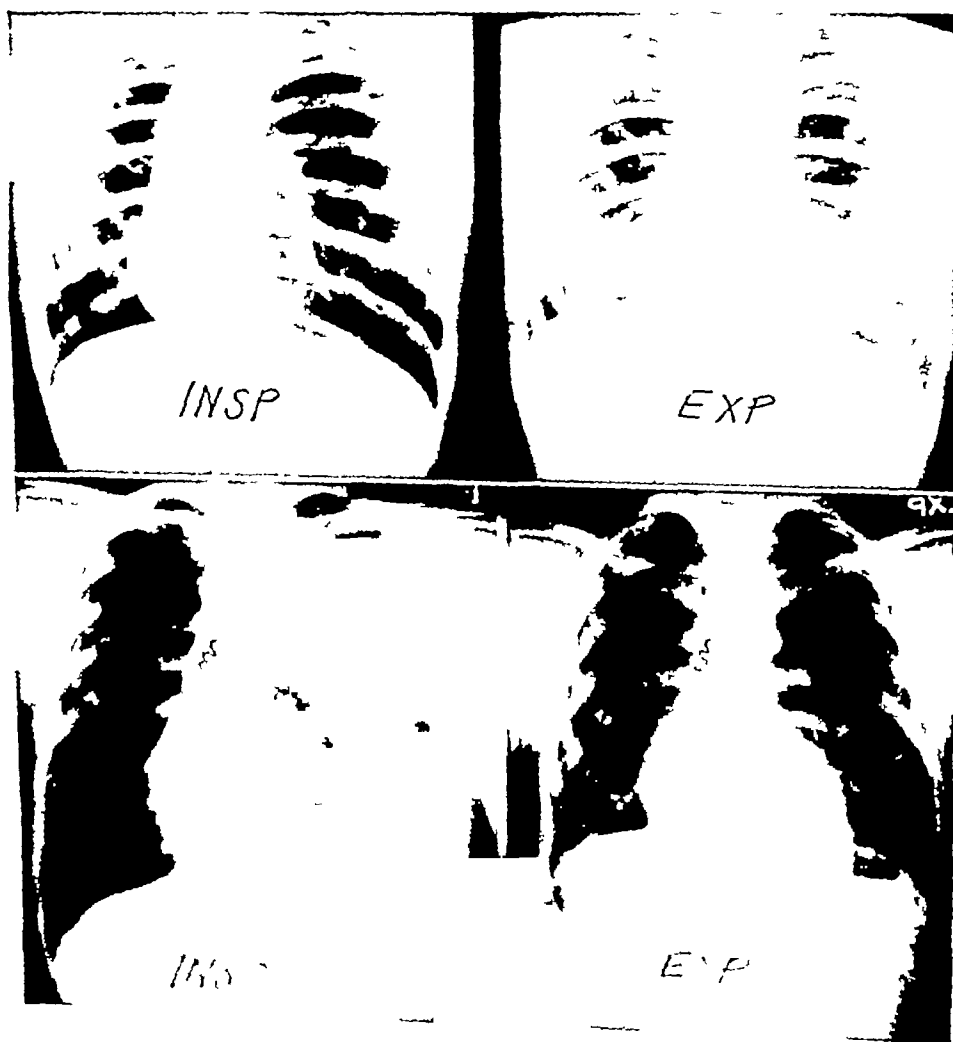
TABLE IIB SUMMARY OF CLINICAL EXPERIENCES INVOLVING MASSIVE TRACHIOBRONCHIAL DERMAL GRAFTS USED WITH RADICAL TOTAL PNEUMONECTOMY FOR EXTENSIVE RIGHT UPPER LOBE CARCINOMA

	AGE	DIAGNOSIS	PROCEDURE	RESULTS
J G	43	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Bronchopleural fistula and empyema healed palliative with distal metastasis
T R	48	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Bronchopleural fistula, empyema and local recurrence—death 5 months postoperatively
N W	49	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Death third postoperative day
G S	54	Extensive right upper lobe carcinoma	Resection of lung, trachea, and carina with graft	Delayed empyema and secondary fistula No recurrence 14 months
G W	59	Extensive right upper lobe carcinoma	Resection of lung trachea and carina with glass prosthesis and pleural graft	Local recurrence and distal metastasis Death 9 months postoperatively

examination revealed a firm fibrous structure of the right main stem bronchus at the level of the orifice of the right upper lobe bronchus. No definite lumen could be visualized. In August 1951 he was operated upon in the free down position through a right high posterolateral incision. Except for some generalized emphysema the right lung appeared to be healthy. The structure area was exposed and a classical Gebauer type of operation carried out. The structure was found to be approximately 6 mm in length and resembled considerably the type of obstruction seen in coarctation of the aorta. A small graft, approximately 2 by 1 cm in size was utilized. Prior to placement of the graft the fibrotic area of stricture was excised down to healthy cartilage and the cut edges of the bronchial mucosa were approximated by interrupted inverted submucosal sutures of No. 0000 arterial silk. Prophylactic tracheotomy was not used and the postoperative recovery was totally uneventful. The area of bronchial repair has not been visualized by bronchoscopy. The patient has been carefully followed for thirty-two months and subjected to periodic cinefluoroscopic and other examinations. There have been no clinical or radiological evidences to suggest any recurrence of stricture. The right

lung is a little smaller than the left so that slight shift of the heart toward the right on inspiration (Fig 1, *B*) is still noted, but this continues progressively to decrease. This patient shows no evidences of decrease in respiratory reserve and his maximum breathing capacity is within normal limits for this age. We feel justified in presuming that the grafted and reconstructed area of the right main stem bronchus has been able to grow in a satisfactory manner with the rest of the tracheobronchial tree.

A



B

Fig. 1.—(A) Frontal view of chest in inspiration and expiration twenty-nine months after operation. (B) Lateral view of chest in inspiration and expiration twenty-nine months after operation. (C) Frontal view of chest in inspiration and expiration thirty-two months after operation. (D) Lateral view of chest in inspiration and expiration thirty-two months after operation. (E) Frontal view of chest in inspiration and expiration thirty-two months after operation. (F) Lateral view of chest in inspiration and expiration thirty-two months after operation.

In December, 1952, a 4-year-old child (L F), was referred to us, again with the impression of "bronchial asthma." This child had had recurrent attacks of pneumonia for a 3½-year period and many episodes of very severe dyspnea. A tendency to very severe dyspnea and wheezing in response to any respiratory tract infection had been noted since birth. The patient presented definite clinical evidence of partial tracheal obstruction. On roentgenological examination (Fig 2), a large mass could be seen to be present between the trachea and esophagus producing considerable compression and distortion of both organs. On Dec 15, 1952, with the patient in the lateral position a high posterolateral incision was made. Preliminary bronchoscopy had not been carried out because of the degree of compression of the trachea known to be present and the fear that any trauma incident to bronchoscopy might produce swelling sufficient to totally occlude the air passages. We



Fig 2—(L F) Preoperative x-ray studies demonstrating posterior tracheal mass producing major compression and distortion of both esophagus and trachea. Postoperative inspiration and expiration studies sixteen months later showed normal pulmonary function and no evidence of recurrence of the original bronchogenic cyst of the posterior tracheal wall.

had not anticipated the need for resection of the posterior tracheal wall or we would have utilized the face down position. The technical difficulty of the procedure was greatly increased by the lateral approach. A Portex endotracheal tube was passed beyond the area of tracheal compression before positioning the patient. Exploration revealed a large firm walled, multilocular cystic mass which later proved to be a bronchogenic cyst. The membranous portion of the tracheal wall was so thinned as to be actually transparent and slight tears in this structure rapidly became major sized defects. The tissue obviously was inadequate and therefore, a classical Gebauer dermal graft, 2.5 cm long by 1 cm wide was placed. Because of considerable chronic bronchopulmonary suppuration a prophylactic tracheotomy was carried out at the conclusion of the procedure. Some adherent exudate formed in the region of the graft requiring bronchoscopy on the second postoperative day.

This procedure was well tolerated. In order to prevent the tracheotomy tube from impinging upon the grafted area, a specially shortened tracheotomy tube was utilized. His postoperative course was otherwise uneventful, the tracheotomy was removed on the fourteenth postoperative day, and he has been totally asymptomatic for the past sixteen months. There has been no recurrence of wheeze or asthmatic behavior. Repeated roentgenographic examination has shown no evidence of tendency to recurrence of the diffuse obstructive emphysema so prominently noted before operative intervention. The child has grown well, and we presume that the reconstructed area of the trachea has grown commensurately.

Adenoma of Carina and Left Main-Stem Bronchus—We have had opportunity to resect adenomas involving the carina on two occasions. The first patient was reported in a previous communication¹⁷ and referred to in the introduction to this publication. The extensive nature of the adenoma throughout the right main stem bronchus, as well as the carina, required that she be treated by a total pneumonectomy and excision of the carina and adjacent tracheal and left bronchial wall. It was possible to close the defect without graft.

In July, 1953, a 52-year-old man presented himself with a history of excessive smoking for thirty-two years, chronic cough, frequent colds without evident suppuration, and a recent rather copious hemoptysis. Investigation revealed that he had been conscious of a wheeze over his left chest in association with respiratory tract infections for the past few months. Such a wheeze was demonstrable on auscultation over the left main-stem bronchus. Roentgenograms revealed large calcified hilar nodes and obstructive emphysema involving the left lung. Laminographic studies (Fig. 3 A) revealed a mass in the upper portion of the left main stem bronchus not completely occluding the orifice. On bronchoscopic examination a typical adenoma was seen occupying the upper medial surface of the left main stem bronchus and extending to the level of the carina. There was no evidence of suppuration distal to the adenoma. On Sept. 14, 1953, operation was performed with the patient in the face-down position utilizing the high left posterolateral approach. It was necessary to mobilize the aortic arch. The posterior wall of the left main-stem bronchus was incised below the level of the adenoma and the tumor removed with an adequate margin. Adequacy of excision was confirmed by a frozen section study of the cut edges. It was necessary to resect the carina and the defect was reconstructed with a 1.5 by 1.75 cm. dermal graft. Pathologic examination revealed a typical adenoma without evidence of carcinomatous degeneration. No postoperative radiation therapy was utilized. The patient's postoperative course was uncomplicated. He has been totally asymptomatic for the past seven months, and roentgenologic studies reveal the left lung with a good ventilatory capacity (Fig. 3 B).

Large Carcinoma With Respiratory Insufficiency—There have been two instances in which patients presented with resectable tumors which had extended a large part or origin in the hilar area of the involved lobe into the main stem bronchus. In both of these patients a severely de-

pleted respiratory reserve forbade any consideration of total pneumonectomy. It was, therefore, decided that the patients should be treated by lobectomy, radical excision of hilar and mediastinal nodes, and removal of a sufficient margin of the adjacent main-stem bronchus to allow eradication of neoplastic tissue. The resultant defect in the bronchial wall would be repaired by an adequate sized dermal graft. The first patient (J. C.) was operated upon in July, 1953. He was shown to have a carcinoma of the right upper lobe bronchus which had extended for a short distance into the lumen of the right



Fig. 2—(S. H.). *A*, Tomographic study demonstrating endobronchial tumor in left main stem bronchus near level of carina. Surgical excision required concomitant removal of the carina for adequate margin beyond a carcinoid type of adenoma. *B*, Postoperative inspiration and expiration films seven months following surgical excision of the adenoma and reconstruction of the bronchial and carinal defect with a Gebauer graft.

main stem bronchus. A roentgenogram (Fig 4, A) revealed that the contralateral or left upper lobe, was replaced with large emphysematous blebs, and there was some generalized hypertrophic emphysema. His maximum breathing capacity was 49 L per minute and it was felt that total right pneumonectomy would produce unjustifiable respiratory limitations. Right upper lobectomy, with resection of the adjacent wall of the trachea and right main-stem bronchus along with radical gland resection was performed. The defect was reconstructed with a rather large graft 5 by 3 cm. over all. Because of the size of the graft it was desired to have constant access to tracheal secretions in a



A

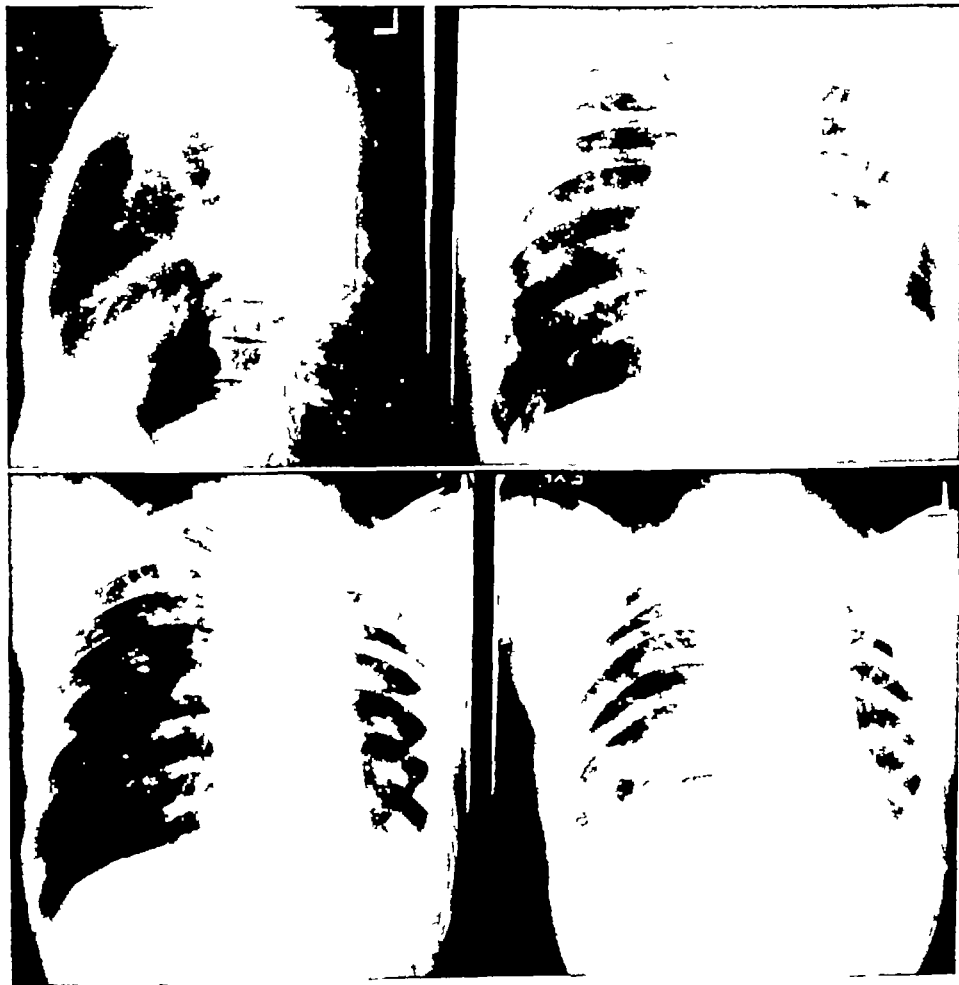


B

FIG. 4. A. Roentgenogram of the chest showing a large mass in the right upper lobe. B. Roentgenogram of the chest showing a smaller mass in the right upper lobe. C. Roentgenogram of the chest showing a smaller mass in the right upper lobe. D. Roentgenogram of the chest showing a smaller mass in the right upper lobe.

patient with respiratory insufficiency, and a prophylactic tracheotomy was carried out. Postoperative course was uneventful, and the tracheotomy was removed on the twelfth postoperative day. A study of the resected lesion revealed the tumor to be quite anaplastic and, therefore, four weeks following operation he was given intensive deep x-ray therapy to the area of the lower trachea, right main stem bronchus, and mediastinal node region. The patient has been totally asymptomatic for the ensuing nine months. There has been no evidence of recurrence of the disease. Roentgenological examination

A



B

FIG. 7.—(A, B). A. Preoperative chest roentgenogram showing extensive bilateral pulmonary fibrosis and complete atelectasis of the left lower lobe. Broncho copy revealed an extensive bronchoalveolar carcinoma of the left lower lobe bronchus. B. Postoperative inspiration and expiration roentgenographic studies five months after surgery demonstrating satisfactory ventilatory function after plastic reconstruction of left upper lobe and left main stem bronchus.

(Fig 4 B) would suggest that there has been no progression of the hyper-trophic emphysema noted in the right middle and lower lobes prior to operative intervention.

In November, 1953, a 58 year old man (P II) was found to have carcinoma arising in the region of a long standing area of bronchostenosis in the left lower lobe bronchus. The neoplastic process had extended upward and medially along the bronchial wall to a level parallel to the orifice of the left upper lobe bronchus. Severe associated chronic pulmonary disease, consisting of intensive pulmonary fibrosis and emphysema (Fig 5, A) with a markedly decreased maximum breathing capacity, precluded any consideration of total pneumonectomy. Therefore on Nov 16 1953 utilizing a left lateral approach, the entire left lower lobe hilar and mediastinal nodes, and an adequate margin of the left main stem bronchus were resected. A graft 35 cm long and approximately 2 cm wide was utilized to reconstruct the airway into the left upper lobe bronchus. No major evidence of suppurative tracheobronchial disease was present and because only a moderately large graft had been placed prophylactic trichotomy was not instituted. Because of the severe degree of pulmonary fibrosis postoperative x-ray therapy was not given. The patient has been active without evidence of recurrence of disease, for five months. Periodic fluoroscopic and physical examinations have indicated satisfactory ventilatory capacity of the remaining lung tissue on the left (Fig 5, B).



Fig 5. (A) Frontal view of chest showing large tumor in right upper lobe. (B) Frontal view of chest showing large tumor in right upper lobe. (C) Frontal view of chest showing large tumor in right upper lobe. (D) Frontal view of chest showing large tumor in right upper lobe.

Left Lower Bronchogenic Carcinoma Arising in the Region of the Right Upper Lobe Orifice. We have had opportunity to carry out total right pneumonectomy associated radical mediastinal gland excision and concomitant tracheal resection of the adjacent tracheal wall tumor and medial wall of left bronchus in two patients. The resultant defect was replaced by a tracheal graft. In one patient in whom the excisional

defect measured approximately 10 cm in length, a flanged Pyrex glass tube (Fig 6) was used for reconstruction. Absorbable sutures were placed about the flanged area and brought out through the tracheobronchial wall at three points, both above and below the defect, to keep the prosthesis in place. A double thickness pedicle pleural graft was then sutured over the defect so as to make it airtight. Prophylactic tracheotomy was not performed. This operation was carried out in May, 1948, and the hazards of removal of such a prosthesis had not been described. Two months following surgery the patient was readmitted to the hospital and, through a cervical tracheostomy, the prosthesis was removed. Stenosis of the reconstructed area slowly but progressively ensued. The patient succumbed nine months after initial operative procedure due to local and generalized recurrence of carcinoma. During the initial two months following surgery, when the prosthesis was still in place, the patient had been distinctly palliated. Following removal of the prosthesis and progressive bronchial stenosis the patient was essentially totally debilitated. It is now felt that the tube could safely have been left in place and the palliation and temporary rehabilitation would have fully justified the operative procedure. Following our experiences with this patient, we awaited the advent of the Gebauer dermal graft before subjecting further similar patients to this type of operative procedure. During the past fourteen months, we have had opportunity to operate upon four additional patients with extensive carcinoma of this type. The first such patient (G S) was operated upon and the defect reconstructed with a graft 6 cm in length and 3 cm in width. Prophylactic tracheotomy was not instituted. The patient's postoperative course was uneventful. Constant major attention was paid to the positioning of the mediastinum and to the pressures presenting in the right pleural space. An attempt was made to try to keep the mediastinum slightly to the left of the midline. Not long after his discharge from the hospital the patient began to run a low grade fever. He lived at a considerable distance and did not return to us for further evaluation until three months after the initial operative procedure. By this time an extensive empyema of the right pleural space had been produced and could not be controlled by aspiration measures. This intrapleural infection produced a progressive and essentially complete necrosis of the grafted area with resultant broncho-pleural fistula. External drainage was necessary at which time a foreign body was discovered in the pleural space and removed. The patient has been maintained on adequate drainage and despite the metastatic involvement of deep mediastinal nodes removed at the time of surgery he now presents no evidence of recurrence of disease fourteen months after the initial procedure. It is our plan to carry out a modified Roberts' type thoracoplasty in the near future. A tantalum mesh sheet will be incorporated in the area of persistent fistula over which will be placed the overlying intercostal muscle bundles. We feel confident that this patient would have had major rehabilitation along with possible eradication of his disease had he not been subjected to a delayed and unnecessary empyema.

Twelve months ago a 43 year old male veteran (J G) with a similar right upper lobe carcinoma was operated upon, and a graft approximately 5

by 3 cm was utilized. He developed a small bronchopleural fistula six days following operation and was treated by thoracotomy drainage and tracheotomy. Two months later the empyema space and fistula were eradicated by means of a Roberts type of thoracoplasty, plus tantalum mesh coverage of the fistulous area. The patient responded well to this operative procedure and, for five months, appeared to have major benefit. He then presented with a large abdominal mass representing a massive metastatic involvement of one kidney. He was given intensive radiation and is still surviving, with considerable persistent residual tumor.

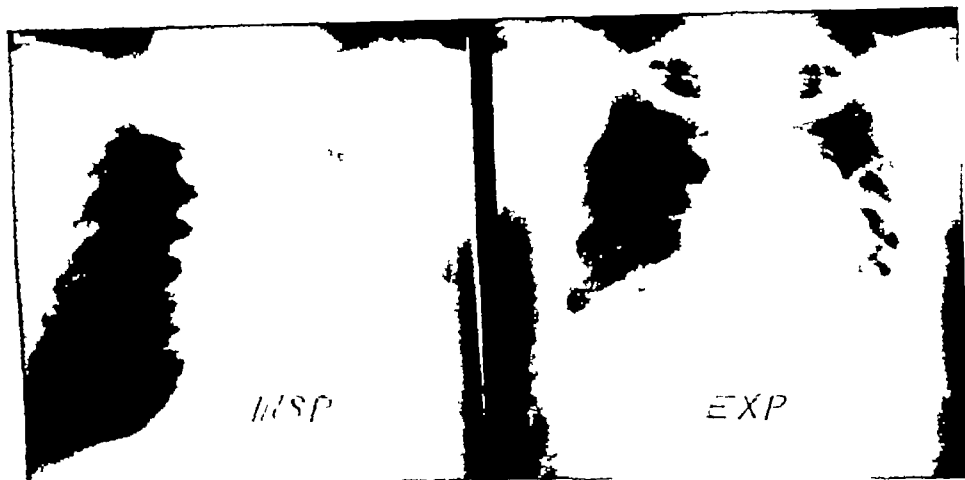


Fig. 7.—(N. T. W.) Inspiration and expiration roentgenographic study to demonstrate complete atelectasis of right lung, marked shift of mediastinum and heart to the right and associated obstructive emphysema of the left lung. (Note: Films printed in reverse.)

Another of these extensive right upper lobe carcinoma patients deserves brief mention. The patient (N. T. W.), a 49-year-old man, presented himself in March, 1954, with a history of cough, hemoptysis, weight loss, and dyspnea. Radiological examination (Fig. 7) showed a very severe degree of obstructive emphysema bilaterally with almost no air exchange in the right lung. Bronchoscopy revealed the right main stem bronchus to be totally occluded with a neoplastic process spreading across the carina on to the medial wall of the left main stem bronchus (Fig. 8). The functional opening into the left main stem bronchus would have admitted approximately two paper matches only. Operation was performed at which time it was not possible to get the long tracheotomy tube into the left main stem bronchus prior to positioning the patient, but an adequate air exchange was maintained with high positive pressure. On opening the chest and slight manipulation of the lung, immediate severe restriction of ventilation in the left lung occurred. A left thoracotomy could be maintained while the thoracic trachea could be exposed and tracheostomy performed, and a Portex intratracheal tube (Fig. 9) introduced past the occluding neoplastic tissue on the left. A radical resection procedure, including total pneumonectomy, radical mediastinal resection, and a left thoracotomy to excise not only the carcinoma and medial wall

Fig 8



Fig 9

Fig 8—(N. T. W.) Resected carina and right lung showing gross extension of tumor into left main bronchus and obstruction of right main bronchus by an extensive bronchogenic carcinoma arising from the orifice of the right upper lobe. The two upper clamps grasp the edges of the left main stem bronchus.

Fig 9—(N. T. W.) Photograph taken during surgery to demonstrate that the operating table Portex tube has been inserted through lateral wall of trachea directly into the left main bronchus to maintain airway while right lung and carina are removed.

left main stem bronchus was performed. A large graft (Fig 10) was utilized to reconstruct the defect utilizing No. 26 silver wire which provided much more adequate support. A very small strand of lower trachea and left main stem bronchial tissue persisted so that approximately 75 per cent of the airway had to be reconstructed. A Portex tube was used for the prosthesis and sewn in place. This type of prosthesis proved to be deficient in two main categories: (1) the thickness of its wall too greatly decreases the functioning lumen; (2) the rigidity of the wall is such as to produce a major tendency for

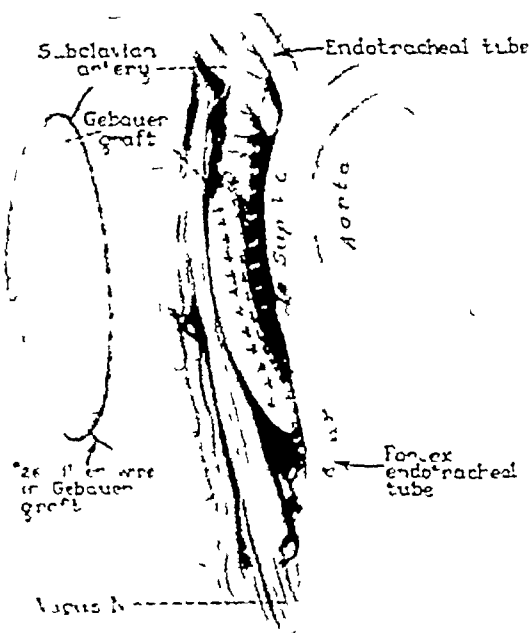


Fig 10.—Artist's drawing which demonstrates the size of the area of the trachea, carina and the left wall of the left main stem bronchus excised with radical right total pneumectomy. The line in the left margin indicates the size and shape of the Gebauer type dermal graft. Note that No. 24 or No. 26 silver wire is used in grafts and placing area is previously excised by extracorporeal means.

the upper portion of the tube to lie against the right lateral wall of the trachea above the defect and thus produce obstruction. This patient had considerable repetitive obstructive phenomena during the first few hours following surgery and this was overcome by placing a No. 4 Portex endotracheal tube into the lumen of the upper stump of the prosthesis through the tracheotomy opening. He had been made immediately following the operative procedure. The patient showed no further improvement but died suddenly on the third postoperative day. Postmortem examination could not be obtained. Death was attributed to accumulated secretions with obstruction. At the time of surgery the possibility of these measurements were made and consideration was given to the possibility of leaving the stump of the left main stem bronchus upward and the upper portion of the tube. This would have involved an up and dis-

placement of over 10 cm and appeared to be totally nonfeasible. We feel that this patient was very instructive. He did show a reasonable method of management of the airway during operation. He further showed some of the dangers of an inadequate prosthesis. A curved polyethylene tube with thin wall would have been preferable. If a Portex tube had been utilized, he should have kept the long tube which is used for initial reconstructive procedures and, at the end of the operation, had it brought out through a tracheostomy opening so that it simulated a long plastic tracheotomy tube capable of spanning the defect. This method was used on one other patient and appeared to show some definite usefulness. The type of apparatus used on this final patient (T. R.) a 48 year old man is illustrated in Fig. 11. This patient had a long graft 75 cm in length, but only 25 cm in width.

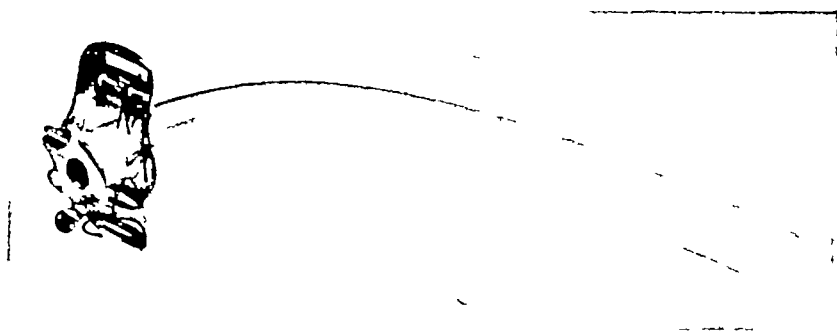


Fig. 11—(T. R.) Portex tube with tracheotomy tube guard and inner tube used through tracheostomy to provide temporary support for graft and to maintain an adequate airway capable of removal, cleansing and reinsertion.

This patient developed a small bronchopleural fistula requiring external drainage with some loss of the graft substance. He had progressive difficulty with narrowing of the main-stem bronchus beginning approximately three months after surgery, and he succumbed five months after surgery because of major local and general recurrence of the disease.

ANESTHETIC AND TECHNICAL CONSIDERATIONS

The major considerations in the line of anesthesia, of course consist of adequate oxygenation and ventilation. For this purpose the patient should be initially intubated with the extra long intratracheal tube. A Portex tube without cuff is adequate if it is of sufficient size to fit snugly into the left main-stem bronchus. On some occasions we have utilized a long McGill catheter with a balloon. One must always have an extra long intratracheal tube at hand which has been sterilized and can be placed through a transthecal tracheostomy at any time indicated. It is mandatory that such an intratracheal tube be available at all times. This tube may be used as a temporary measure and connected directly to the anesthetic machine. While this transthecal tube is being utilized the original catheter may be inserted by the anesthetist and then passed across the defect under direct vision.

by the operator. This catheter may be left in place after complete repair of the defect and closure of the chest. A tracheotomy is then performed. The Portex tube is transected at a height sufficient to allow it to be brought out through the tracheotomy wound which allows us to have produced a long, removable tracheotomy tube made of pliable material which passes beyond the area of defect. It is not thought that this type of prosthesis is necessary except in major-sized grafts. It is our impression that grafts 5 cm. in length or shorter, with adequate intrinsic support, do not necessarily need prosthetic supports. We would also like to emphasize the importance of the supporting sutures about the trachea and bronchus which were emphasized by us in an earlier communication and which are again illustrated in Fig 12. This maintains a good support of the mediastinum and helps to hold open the airway,



Fig 12—Photograph taken during operation which demonstrates (1) the supporting sutures in the trachea and bronchus used to stabilize the mediastinum, and (2) the Portex tube in place maintaining the airway during grafting procedure.

particularly while manipulations such as changing or moving the long intra-tracheal tubes are being carried out. We have found a very marked advantage in utilizing the Rand-Wolfe Respirator, since if high pressures are instituted with this machine for example 20 to 25 cm. of water, adequate aeration of the contralateral lung can be carried out even in the face of definite large bronchial leaks. The delivered pressure to the lung parenchyma is much below the recorded pressure on the machine due to these leaks. The pressure is graduated according to the position of the mediastinum and the patient's needs. In regard to tracheotomy as a postoperative procedure this is decided fundamentally by the amount of suppuration present in the individual patient and by the size and character of the graft utilized.

In regard to technical operative considerations we certainly concur very fully with Gebauer¹⁴ in the benefits of the face down position in the man-

agement of lesions of the posterior tracheal wall and of the left main stem bronchus. The lateral position is quite satisfactory for the radical total pneumonectomy and lobectomy procedures. The preparation of the graft must assiduously follow the detail outlined by Gebauer. We have found it very useful to have a moderate-sized block of balsa wood, which facilitates the anchoring of the graft with the straight pins. We would like to emphasize strongly the need for a paper pattern to be sterile and available. This is placed about the endotracheal tube or prosthesis so that adequate graft to cover the curved contour will be produced. It is very important that the graft be cut distinctly larger than the paper pattern. The paper pattern should be kept in the patient's chart to aid in further investigative study. It is important to stress in the radical pneumonectomy group where approximately three fourths of the circumference of the trachea and bronchus is being removed that very adequate support of the wall of the graft is mandatory. Our experience has shown us that while we agree with Gebauer in the desirability for relatively fine silver wire supporting sutures in the graft when repairing stenotic lesions and just replacing the soft membranous portion of the trachea or bronchus, a considerably different situation is presented when we are replacing major sized areas of trachea or bronchus previously supported by a cartilaginous framework. For this latter purpose, we prefer to use a No. 26 or even a No. 24 silver wire suture for the internal support of a considerably thick walled graft. In the repair of stenotic lesions and replacement of noncartilaginous tracheal or bronchial tissue, the No. 28 or No. 30 silver wire is fully adequate. In the repair of stenotic lesions it is necessary to utilize molds to curve the graft properly. The replacement of major portions of the cartilaginous tracheal wall utilized the indwelling tracheal tube for a prosthesis in place of the mold to aid in the proper curvature of the replacement tissue. Probably the most common error which we have incurred is to obtain grafts of inadequate width. It is important to plan a graft which if it overlies a tube or prosthesis will have some definite redundancy. Compression upon the graft by a prosthesis permanent or temporary produces compression phenomena unfavorable to the circulation. Furthermore a graft which is too snug has a marked tendency to flatten and produce an inadequate tracheobronchial lumen. It is obvious from the preceding discussion that we do not feel that a completely adequate prosthetic substance is at hand and further measures must be carried out to study this problem. Our present preference is for a thin curved polyethylene tube. It is necessary for such a tube to have a distinct difference in the diameters of its upper and lower orifices. We have on all occasions utilized a large pedicle pleural graft which is sewn over the dermal graft or over an exposed prosthesis in one instance. This is necessary in order to seal the airway. We initially used to place Gelform between the pleural and dermal graft but now feel that this decreases potential blood supply for larger grafts. Grafts 3 cm. in length or smaller are carried out in the classical Gebauer manner with the interspersed Gelform. Grafts of greater length and particularly grafts of greater width are felt to require a source of external blood supply and we prefer to employ a pedicle graft utilizing the pleura and fat from the pericardial region because of its

vascularity. Following completion of the graft the pleural cavity is filled with saline to test the pleura and ensure the presence of a completely airtight reconstruction. In cases of total pneumonectomy a thoracotomy tube is placed through the third anterior interspace. This tube is not utilized for drainage, but is clamped an adequate distance from the chest and the clamp handles seal-closed with adequate adhesive strapping. This tube is utilized for frequent measurements of the intrapleural pressures and, if necessary, to serve as a source for injection of either air or plasma to help splint the mediastinum in the proper position.

We do not feel that there need be any major fear of bronchoscopic maneuvers following operative procedures. They have been done frequently with our grafts and have been reported by others. They are useful for the removal of exudate, debris, and granulation tissue should it occur. Certainly bronchoscopy is not justified in these procedures except upon therapeutic indications. If a prophylactic tracheotomy is in place, this opening is utilized for the bronchoscopic approach. If bronchoscopy is required following surgery and the patient does not have prophylactic tracheotomy, it is usually felt advisable to institute the tracheotomy at that time.

FUNDAMENTAL PROBLEMS

There are certain fundamental problems which have been brought forward by experiences in this field. The first is whether or not the procedure is justified. Our experience indicates that it is fully justified in all the circumstances initially set down, with the reservation that there is some doubt relative to its justification in extensive right upper lobe carcinomas which have invaded beyond and across the carina. It is our feeling that, despite our present major complication and minimal palliation rate outlined in our above experiences with these lesions, an open mind should be kept. Further investigative experiences of laboratory and clinical types should be studied. It is our impression that if we will insist upon a more adequate width of graft and the firmer support presented by No. 24 silver wire, some of the complications inherent in the placement of massive grafts in the reconstruction of cartilaginous areas of the tracheobronchial wall will be eradicated. There remains a distinct problem in regard to how large a graft one can place before too high an incidence of partial necrosis and fistula is produced. It is our present feeling that grafts 5 cm. in length and 2.5 cm. in width have been proved to survive, while those over 5 cm. in length, with a width of 3 cm. or greater, stand a high chance of local necrosis. Therefore, in the larger grafts an underlying prosthesis should be utilized. The exact type of prosthesis is not as yet defined but our present trend is toward a specially constructed and perforated polyethylene tube.

The problem of the need of postoperative tracheotomy is also an important question. Certainly there is some benefit in decreasing the pressure produced upon the suture line by cough with the closed glottis and intact cervical tracheal wall. The tracheotomy does distinctly introduce a certain hazard of potential infection of the graft and restricts the normal humidification of the tracheobronchial airway. Our feeling is that the need for tracheotomy is

an individual factor to be determined by graft size, the presence or absence of a prosthesis, and the suppurative aspect of the patient's tracheobronchial tree

Initial experiences with employment of grafts in children suggest strongly that small or moderate-sized grafts will grow. One would fear the use of grafts larger than 3 by 1.5 cm. in the younger age group. Further observation and study, particularly with experimental investigation, are needed to define the fate of grafts placed in infancy or childhood.

A further important problem is the consideration of bringing an adequate blood supply to the base of large grafts. Graft survival requirements imply restrictions produced by dimensions and by the blood supply of the remaining strand of tracheobronchial tissue to which it is attached. It would seem reasonable to hope that a fairly vascular, pedicle type, pleural fat graft, placed directly upon the base of the transplanted dermis, might decrease tendencies to local necrosis of the graft substance.

SUMMARY AND CONCLUSIONS

The background investigations and clinical experiences relative to tracheobronchial reconstruction have been outlined. The value of the contributions of Gebauer, relative to reconstruction of the tracheobronchial tree by grafting and other methods, is emphasized. The indications for tracheal or bronchial reconstruction by grafting procedures have been detailed and fall into three major categories. These consist of the management of acute trauma, of various types of bronchial stenosis, and of neoplastic lesions. Congenital defects may constitute a fourth indication. Experiences with the application of various types of grafting procedures in ten nontuberculous lesions are outlined. Limited experience suggests that grafts are well tolerated in childhood and appear to grow in a satisfactory manner with the child. The use of grafts in the management of bronchogenic cyst of the tracheal wall and of a stricture secondary to prolonged endobronchial trauma by a foreign body have each been carried out successfully. Experiences in two instances show that a more adequate operation for carcinoma arising in the hilar area of a lobe in patients with a severe associated restricted respiratory capacity can be performed. In one instance the graft has successfully tolerated intensive postoperative deep x-ray therapy. An additional experience of management of an adenoma of the left main-stem bronchus and carina, with correction by local resection and grafting, has been presented. It is hoped that the use of bronchial grafts may extend the limits of operability of carcinoma arising in the right upper lobe area so a more adequate excision of tracheal, carinal, and contralateral bronchial tissue may be performed. The limitations and problems inherent in the procedure have been presented.

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(For Discussion see page 252)

BRONCHIAL ANASTOMOSIS AND BRONCHOPLASTIC PROCEDURES IN THE INTEREST OF PRESERVATION OF LUNG TISSUE

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BRONCHIAL anastomosis and bronchoplastic procedures in the interest of preservation of lung tissue are relatively new developments in the field of thoracic surgery. A traumatic, inflammatory, or neoplastic lesion of the bronchus does not always require resection of all of the lung tissue supplied by the involved bronchus. By means of various types of repair, it is possible to excise a portion of bronchus and to restore continuity, thus salvaging a portion or all of the lung tissue distal to the point of excision.

The reports of Gebauer^{1, 2} made before this association have demonstrated very well the merit of the concept of pulmonary salvage through bronchial reparative procedures, particularly with reference to tuberculous broncho stenosis but including bronchial tumors. Many others through individual published reports and discussion of Dr. Gebauer's papers have recorded the successful application of various reconstructive procedures of the bronchus following excision of tumors, traumatic strictures, and accidental bronchial division.

The authors have used a variety of procedures to restore bronchial continuity and preserve good lung tissue in sixteen patients. The bronchial lesions so treated include traumatic occlusion (2), acute traumatic rupture (2), tuberculous stenosis (3), adenoma (2), and carcinoma (7). The individual cases are summarized in Tables I, II, III, and IV with appropriate illustrations.

TABLE I. TRAUMATIC BRONCHIAL RUPTURE

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
1 I M	M 22	Traumatic bronchial occlusion, right main bronchus, 7 weeks following injury	Plastic reconstruction of right main bronchus by means of dermal graft (Fig 1)	July 2, 1949	Bronchus patent Lung fully expanded
2 B S	M 21	Traumatic bronchial rupture, left main bronchus, 6 weeks following injury	End to end anastomosis of left main bronchus (Fig 2)	Aug 8, 1951	Bronchus healed Lung fully expanded
3 J C	M 19	Traumatic bronchial rupture, 6 hours following injury	End to end anastomosis, left lower lobe bronchus to left main bronchus (Fig 3)	April 19, 1951	Bronchus healed Lung fully expanded
4 B H	M 23	Traumatic bronchial rupture, 2 hours following injury	Tracheotomy Suture of tear involving right main, intermediary and superior segmental bronchi (Fig 4)	Jan 20, 1954	Bronchus healed Lung fully expanded

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TRAUMA

Rupture of the bronchus may result from nonpenetrating trauma of the chest. The diagnosis during the period immediately following the injury can be readily made if one keeps in mind the possibility of a tear of the bronchus in a patient who has sustained nonpenetrating trauma of the chest and in whom a tension pneumothorax and/or mediastinal emphysema rapidly develops. A bronchial tear should be suspected if, following decompression of the tension pneumothorax, air continues to bubble vigorously through the water seal system and if the lung fails to expand. Bronchoscopy done immediately following the injury will reveal in most cases the site of the bronchial tear, as usually it occurs in either main bronchus at or within a few centimeters of the carina. However, secondary bronchi may be torn. There may be either a partial tear or complete transection of the bronchus. A great many of these injuries have ended fatally within a short time following the accident because of failure to make the diagnosis until autopsy revealed the true condition.

Prompt recognition of a traumatic bronchial rupture *must be followed* immediately by thoracotomy and repair of the torn bronchus as illustrated in Cases 3 and 4. By means of an end-to-end anastomosis the left lower lobe was preserved in Case 3. The suture of a large tear involving the right main, intermediary and superior segmental bronchi resulted in the salvage of an entire lung in Case 4.

If the patient survives the early posttraumatic period the unrepaired ruptured bronchus heals with either complete occlusion or stricture of the bronchus. Bronchial anastomosis is indicated for complete traumatic bronchial occlusion should such a patient be seen months or even years after the initial injury. Pneumonectomy is not justified unless pulmonary suppuration is present, and this cannot occur in the case of complete occlusion.^{4, 5} The collection of thick viscid mucus within the bronchi of the lung blocked by complete bronchial occlusion must not be mistaken for suppuration. The lung itself although atelectatic, undergoes no pathologic change. A bronchoplastic procedure is carried out in Case 1 or an end-to-end anastomosis as made in Case 2 should be done to relieve the bronchial occlusion and restore bronchial continuity. The atelectatic lung will reexpand and resume function if bronchial continuity is restored, even years after the initial injury. Samson and Evans⁶ have recently successfully reexpanded a lung by means of a bronchial anastomosis in a patient fifteen years after a traumatic bronchial occlusion was sustained.

The pulmonary suppuration which will develop if the bronchial tear heals by stricture formation without complete occlusion makes imperative the early diagnosis and resection of the traumatic bronchial stricture and restoration of bronchial continuity by end to end anastomosis.

By early diagnosis and bronchial reconstructive procedures, the mortality of bronchial rupture can be markedly reduced and lung function preserved.

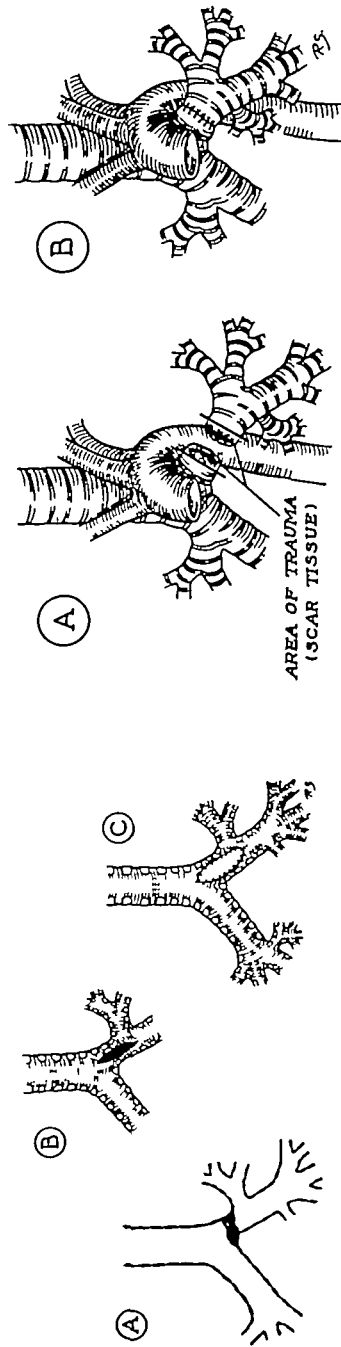


Fig 1

Figs 1 4—Traumatic bronchial rupture

Fig 1—Bronchial occlusion right main bronchus Incision and dermal graft seven weeks after injury (Case 1)
Fig 2—Complete separation of left main bronchus Excision of scar and end to end anastomosis six weeks after injury (Case 2)

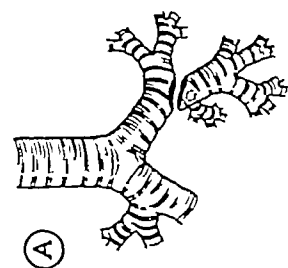
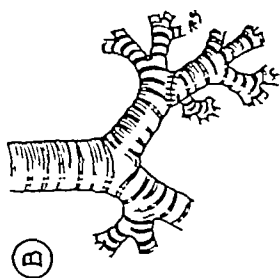
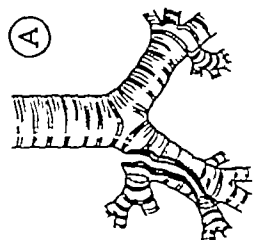
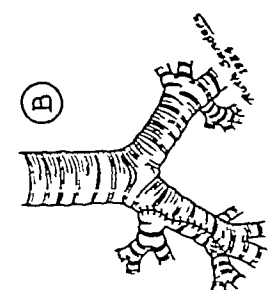


Fig. 1

Fig. 3

Fig. 3—Complete separation of left lower lobe bronchus. End to end anastomosis six hours after injury (Case 3)
 Fig. 4—Tear of right main intermediary and superior segmental bronchi. Suture two hours following injury

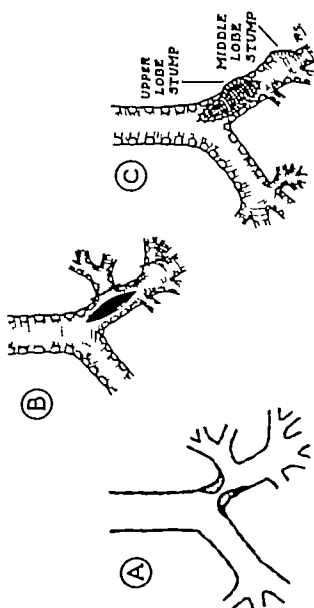


Fig 5

Figs 5 and 6—Tuberculous bronchostenosis

Fig 5—Healed stenosis right main bronchus. Right upper and middle lobectomies. Incision of stenotic area and reconstruction of dermal graft reinforced by steel wire mesh (Case 5)

Figs 6—Healed stenosis right main and right upper bronchi. Resection of right main bronchus and right upper lobe end-to-end anastomosis (Cases 6 and 7)

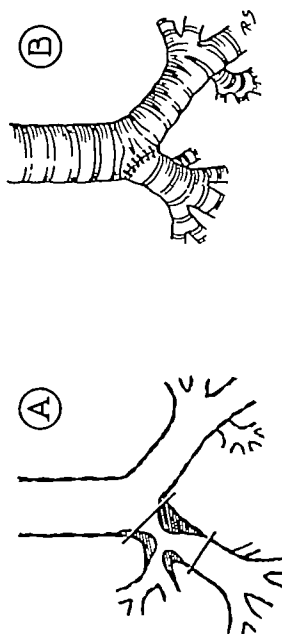


Fig 6

TUBERCULOUS BRONCHOSTENOSIS

Healed tuberculous bronchostenosis, usually involving the primary and secondary bronchi, may be resected and bronchial anastomoses done. If the stenosis involves the main bronchus and the lower trachea, as in Case 5, a plastic reconstruction by means of a dermal graft reinforced by steel wire mesh may be utilized after incising the stenotic area. A lobectomy or segmental resection may be done at the same time as the bronchial anastomosis or plastic reconstruction. By establishing bronchial continuity the remaining healthy lobes of lung are preserved as illustrated in Cases 5, 6 and 7. By means of these procedures a total of 5 lobes of lung were preserved in these 3 cases. There were no complications and no mortalities.

TABLE II TUBERCULOUS BRONCHOSTENOSIS

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
5 A T	F 20	Pulmonary tuberculosis with 2 cavities persisting following thoracoplasty in 1950. Healed stenosis, right main bronchus.	Right upper and right middle lobectomies. Plastic reconstruction of right main bronchus and lower trachea by means of dermal graft reinforced by steel wire mesh (Fig 5).	Dec 15, 1951	Bronchus healed. Good expansion of lower lobe. Sputum negative.
6 D C	F 34	Pulmonary tuberculosis, right upper lobe. Healed stenosis, right main and right upper lobe bronchi.	Resection of right upper lobe and right main bronchus. End to end anastomosis of intermediate bronchus to trachea (Fig 6).	July 30, 1953	Bronchus healed. Right middle and lower lobes fully expanded. Sputum negative.
7 R B	F 31	Pulmonary tuberculosis with destruction of right upper lobe.	Right upper lobectomy which compromised the lumen of the right main bronchus. Transverse division of bronchus and end to end anastomosis.	Nov 7, 1953	Bronchus healed. Right middle and lower lobes fully expanded. Sputum negative.

TABLE III BRONCHIAL ADENOMA

CASE	SEX AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
8 J S	F 24	Bronchial adenoma medial wall of right main bronchus.	Wedge excision and transverse suture (Fig 7).	June 12, 1951	Bronchus healed. Lung fully expanded. Well to date.
9 J H	M 13	Bronchial adenoma anterolateral wall left lower lobe bronchus.	Left lower lobectomy and excision of adenoma, preserving a flap of bronchial wall used for reconstruction (Fig 8).	June 2, 1952	Bronchus healed. Left upper lobe fully expanded. Well to date.

BRONCHIAL ADENOMA

Because bronchial adenoma is a tumor of low-grade malignancy it lends itself well to localized resection and bronchial reconstructive procedures in the interest of preservation of healthy lung tissue. Only in the presence of distal pulmonary suppuration is the resection of lung tissue justified. Case 8

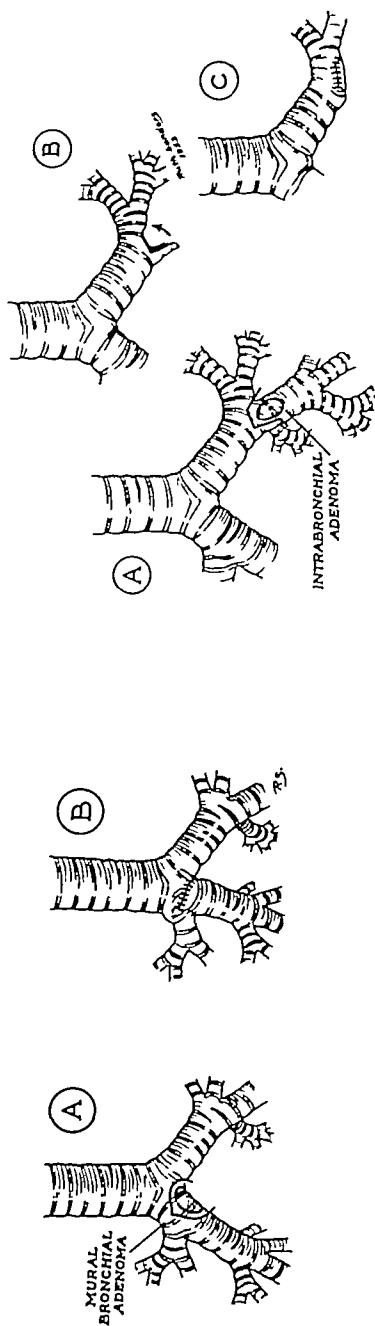


Fig. 7

Figs 7 and 8—Bronchial adenoma.

Fig. 7—Mural adenoma right main bronchus. Wedge excision and transverse suture (Case 8)

Fig. 8—Intrabronchial adenoma left lower lobe bronchus. Left lower lobe bronchus. Transverse lobectomy and excision of adenoma preserving a flap of bronchial wall for reconstruction (Case 9)

illustrates the preservation of an entire right lung which was healthy in spite of an adenoma in the medial wall of the right main bronchus. Wedge resection and transverse suture resulted in complete excision of the tumor with restoration of bronchial continuity.

Bronchial adenomas may be so situated that a tumor in addition to producing suppuration in the lobe distal to it has grown so close to another bronchial orifice as to indicate a wider bronchial resection to effect a cure. In such cases it is unnecessary to sacrifice the healthy lung tissue because a bronchoplastic procedure or bronchial anastomosis may be carried out which will permit wide excision of the tumor and yet preserve the healthy lobe. By means of a flap of bronchial wall it was possible to preserve the left upper lobe in Case 9. Pneumonectomy and loss of function would otherwise have been necessary for adequate removal of the adenoma.

BRONCHOGENIC CARCINOMA

The authors are of the opinion that bronchial reconstructive procedures have a definite place in the surgery of bronchogenic carcinoma under certain limited conditions. By means of transverse suture or dermal grafts it is possible to restore bronchial continuity following resection of an entire lung, the carina, and a portion of the opposite main bronchus for a bronchogenic carcinoma. This procedure as suggested by Abbott,⁷ extends the operability of certain lesions which impinge upon the carina and trachea as illustrated in Case 10. In certain other instances pulmonary function may be diminished to the degree that it prohibits the resection of an entire lung but will permit the resection of a lobe. By means of a limited resection and bronchial anastomosis the removal of a carcinoma involving the right main bronchus was made possible in Case 15. Pneumonectomy was out of the question because a contralateral thoracoplasty had been done seven years before for tuberculosis. In two other cases (Cases 11 and 12) lobectomy with wedge resection and bronchial anastomosis were indicated because of poor pulmonary function. In three patients (Cases 13, 14, and 16) a lobectomy and bronchial anastomosis were done deliberately on the premise that these procedures were adequate operations for these particular carcinomas.

Of the seven patients in whom a bronchial reconstruction was done following resection for bronchogenic carcinoma, a total of 8 lobes of lung were preserved. Three patients have died of carcinoma 4 months, 5 months, and 1 year respectively after the operation. It is interesting that in two of these three patients a limited resection and bronchial anastomosis were done as a compromise with pneumonectomy because of poor pulmonary function. The remaining four patients are alive and well 1, 11, 12, and 18 months respectively following the operation. It is noteworthy that three of these four surviving patients were those in whom limited resections and bronchial anastomoses were done deliberately as an adequate operation for their respective carcinomas.

An empyema developed in two patients following transverse suture. Both responded promptly to treatment. A mediastinal and subcutaneous emphy-

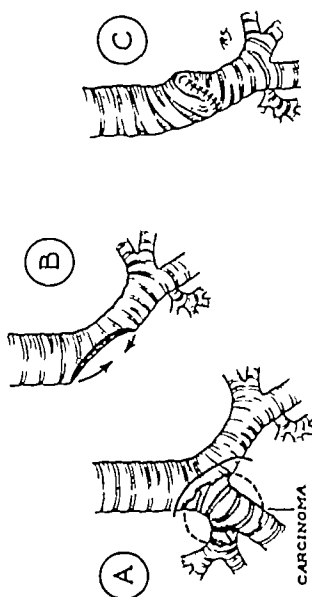


Fig 9

Figs 9 12—Bronchogenic carcinoma

Fig 9—Epidermoid carcinoma of right main bronchus trachea and carina. Resection of right lung lower trachea carina and medial wall of left main bronchus. Reconstruction by transverse suture. (Case 10)

Fig 10—Epidermoid carcinoma right upper lobe bronchus. Right upper lobectomy wedge resection of right main bronchus and transverse suture. (Case 11)

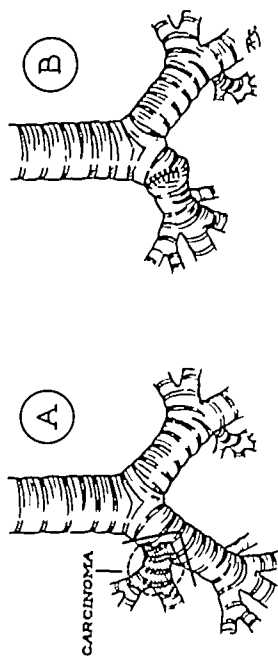


Fig 10

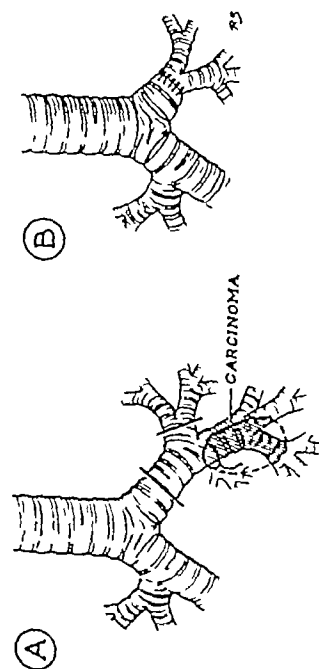


FIG. 11

FIG. 11.—Carcinoma of left upper lobe and left main bronchus (Cases 12, 13 and 14).

FIG. 12.—Carcinoma of right upper lobe and right main bronchus (Case 15).

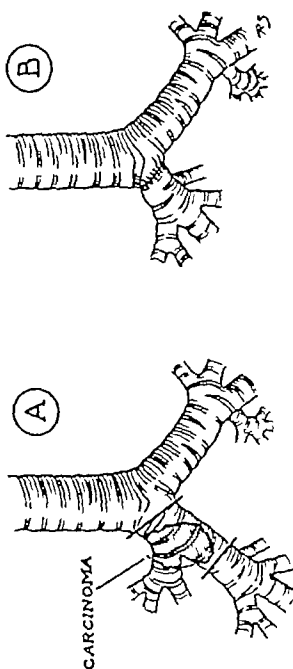


FIG. 12

FIG. 12.—Resection of right main bronchus and right upper lobe end-to-end anastomosis.

FIG. 13.—Resection of left lower lobe and left main bronchus (Case 16).

TABLE IV BRONCHOGENIC CARCINOMA

CASE	SEX	AGE	DIAGNOSIS	SURGICAL PROCEDURE	DATE	RESULT
10 L McC	M	57	Bronchogenic carcinoma, epidermoid, right main bronchus, trachea, and carina, with lymph node metastases	Resection of right lung, lower trachea, carina, and medial wall of left main bronchus Reconstruction of left main bronchus by transverse suture of left main bronchus to trachea (Fig 9)	June 1, 1951	A one year survival. Developed empyema and fistula at site of suture line which healed with drug therapy and drainage Died June 14, 1952, of carcinoma
11 V S	M	56	Bronchogenic carcinoma, epidermoid, right upper lobe bronchus with lymph node metastases Emphysema with poor respiratory function	Right upper lobectomy, wedge resection of right main bronchus and transverse suture (Fig 10)	Nov 11, 1952	Leak at suture line and empyema Healed with drainage by February, 1953 Well to date
12 S S	M	58	Bronchogenic carcinoma, epidermoid, left lower lobe bronchus with lymph node metastases Emphysema with poor respiratory function	Resection left lower lobe and left main bronchus End to end anastomosis left upper lobe and left main bronchus (Fig 11)	April 9, 1953	Bronchus healed and left upper lobe expanded Pelvic metastasis Died Aug 30, 1953, with recurrence of carcinoma in bronchus and mediastinum
13 H E	M	47	Bronchogenic carcinoma, undifferentiated, superior segmental bronchus, left lower lobe, with lymph node metastases	Resection of left lower lobe and portion of left main bronchus End to end anastomosis, left upper lobe and left main bronchus (Fig 11)	April 21, 1953	Bronchus healed Left upper lobe well expanded. Well to date
14 A B	M	50	Bronchogenic carcinoma, epidermoid, superior segmental bronchus, left lower lobe, with metastasis to one lymph node	Resection of left lower lobe and portion of left main bronchus End to end anastomosis of left upper lobe and left main bronchus Tracheotomy (Fig 11)	June 13, 1953	Emphysema relieved by tracheotomy Bronchus healed Left upper lobe well expanded Well to date
15 C F	M	50	Bronchogenic carcinoma, epidermoid, right main bronchus, with metastases to regional lymph nodes Left 8 rib thoracoplasty for tuberculosis, 1946	Resection right main bronchus and right upper lobe End to end anastomosis of intermediary bronchus to trachea Tracheotomy (Fig 12)	June 8, 1953	Bronchus healed Right middle and lower lobes expanded well Died Oct. 16, 1953, of carcinoma in right bronchial tree
16 J K	M	55	Bronchogenic carcinoma, epidermoid, apical posterior bronchus, left upper lobe No node involvement	Left upper lobectomy Left lower lobe bronchus compromised by closure Wedge resection and transverse closure	March 12, 1954 March 16, 1954	Bronchus healed Left lower lobe well expanded Well to date

sema developed immediately postoperatively in one patient (Case 14) due to a leak of air from the bronchial anastomosis and was relieved by tracheotomy. There were no operative mortalities. Recurrence of the carcinoma occurred in the reconstructed bronchial lumen in two patients (Cases 12 and 15). In each instance a localized resection and bronchial anastomosis were done as a compromise with pneumonectomy because of poor respiratory function.

TECHNIQUE

The amazing healing powers of the bronchus have been well demonstrated for years by the simple closures of bronchial stumps used by thoracic surgeons. Experimental and clinical observations with bronchial anastomoses have further substantiated this fact.

No attempts were made to preserve the bronchial vessels in this series of cases. The bronchus was cleanly divided and any bleeding from the bronchial vessels controlled. Careful suturing was done, using interrupted fine cotton or cable wire sutures so placed as to bring the mucosal edges into careful opposition. In so far as possible, the suture was placed through the bronchial wall and submucosa to avoid piercing the mucosa and was tied on the outside of the bronchus. Care was taken to make the closure of the bronchial wall airtight.

Tracheotomy was not done routinely. The bronchial secretions, which were usually troublesome during the first few days postoperatively, were removed in most instances by means of intratracheal catheter and suction. In those instances in which pulmonary function is reduced to an extreme degree a tracheotomy should be done at the time of the operation, as in Case 15. Should an air leak at the site of the suture line develop in the immediate postoperative period a tracheotomy should be done to reduce the intratracheal and intrabronchial pressure.

Postoperative bronchoscopy was avoided in most instances. Such instrumentation early in the postoperative period may traumatize the delicate mucosa at the line of suture or reconstruction. Late bronchoscopy for the purpose of inspection of the anastomotic site is unnecessary unless the patient is having symptoms such as an irritative cough, indicating sutures lying within the lumen of the bronchus. Should this occur, bronchoscopic removal is indicated.

The use of the prone position during any reconstructive procedure involving the bronchus or trachea facilitates the operation considerably from the standpoint of anatomic exposure, anesthesia and maintenance of a clear airway. Because the main bronchi are situated posteriorly, the most direct exposure for most bronchial anastomoses is obtained with the patient lying in the prone position. In this position the patient tolerates an open bronchus for longer periods of time than in the lateral position because of the absence of the need for positive pressure anesthesia. Indeed the patient may respire room air through the bronchotomy opening for short periods of time with no deleterious effects.

The airway remains clear of bloody drainage because any bleeding usually drains out into the pleural cavity rather than into the bronchus.

The passage of the intratracheal tube into the contralateral bronchus is usually unnecessary in the prone position. In the case of extensive resections about the carina, however, this is desirable. In some instances in this series of patients the main bronchus was temporarily closed by means of a soft

clamp, a sponge, or interrupted fine sutures. In one instance (Case 15), a small intratracheal tube was introduced into the distal intermediary bronchus, brought out through the wound, and the two remaining lobes respired by means of a second anesthetic machine.

In the case of bronchogenic carcinoma, the prone position makes possible an extensive mediastinal and pericardial dissection even in those instances in which a lobectomy is done. All lymph-node-bearing areas can be readily approached.

DISCUSSION

There can be no question of the usefulness of bronchial suture, bronchial anastomosis, and bronchoplastic procedures for traumatic bronchial rupture, tuberculous bronchostenosis, and bronchial adenoma. These procedures should be considered in the surgical attack on any benign bronchial lesion to avoid unnecessary sacrifice of good lung tissue.

The utilization of these procedures in the surgical attack on bronchogenic carcinoma must be carefully considered. Inadequate pulmonary reserve and the extension of operability justify their use in certain instances. The authors are of the opinion that, in the interest of preservation of lung tissue and a lower mortality, the operation of lobectomy with wide resection of the carcinoma, detailed dissection of the mediastinum, and bronchial anastomosis may be done deliberately in those cases in which it is believed to be an adequate operation for the tumor.

Our experience with the treatment of bronchogenic carcinoma has indicated that lobectomy, where feasible, yields at least as good results as pneumonectomy, with a lower mortality. The mortality rate in our hands for the 239 resections done for bronchogenic carcinoma during the period from 1945 to 1954 was 10.6 per cent for pneumonectomy and 3.1 per cent for lobectomy (Table V). The survival rates for four years or more for resections done for bronchogenic carcinoma in the years 1945 through 1950 are 25 per cent for lobectomy compared with 13 per cent for pneumonectomy (Table VI). In many instances more favorable lesions, such as the well localized peripheral

TABLE V OPERATIVE MORTALITY FOR BRONCHOGENIC CARCINOMA, 1945-1954

TYPE OF RESECTION	NUMBER	DEATHS	MORTALITY (%)
Pneumonectomy	141	15	10.6
Lobectomy	98	3	3.1
Total	239	18	7.5

TABLE VI SURVIVAL RATES OF PATIENTS WITH BRONCHOGENIC CARCINOMA OPERATED UPON, 1945-1950

TYPE OF RESECTION	NUMBER	SURVIVORS	SURVIVAL RATE (%)
Pneumonectomy	83	11	13.2
Lobectomy	40	10	25.0
Total	123	21	17.0

carcinoma were removed by lobectomy, although this was not entirely true the indications being in some instances chest wall involvement and poor pulmonary or cardiac reserve

Pneumonectomy is the ideal cancer operation in the majority of bronchogenic carcinomas. There remains a group of localized lesions however, for which lobectomy may be a desirable and practical operation because of the lower mortality rate and the advantages resulting from the preservation of lung tissue. In addition to the indications for lobectomy already given, this operation combined with bronchial resection and anastomosis may be considered for the well-localized small, centrally located lesions. Furthermore, this procedure will permit wider excision of the bronchus in those patients in whom limited cardiac or respiratory reserve makes lobectomy imperative.

SUMMARY

1 Sixteen cases involving the use of bronchial anastomosis and bronchoplastic procedures in the interest of preservation of lung tissue are presented

2 These procedures were used successfully for traumatic bronchial rupture (4) tuberculous bronchostenosis (3) and bronchial adenoma (2)

3 Bronchial anastomoses have been utilized for bronchogenic carcinoma in seven patients because of inadequate pulmonary reserve (3) deliberately for small centrally located lesions (3) and to extend operability in one patient. Three patients died of their carcinoma within one year after operation. The remaining four patients in three of whom the operation was done deliberately are alive after 1 11 12 and 18 months respectively.

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ADDENDUM

A total of twelve bronchoplastic procedures with resection for bronchogenic carcinoma have now been performed by the authors. The indications were inadequate pulmonary reserve in four deliberately for centrally located lesions in six and to extend operability in two patients. One operative mortality occurred twelve days after an end to end anastomosis of the left main bronchus and trachea following resection of the right lung and carina. Death was due to inadequate pulmonary function. Three patients died of carcinoma within one year of the operation. The remaining eight patients are alive and well for varying periods up to twenty six months following operation.

DISCUSSION

(PAPERS BY ABBOTT, VANFLEIT, AND ROBERTO, AND PAULSON AND SHAW)

DR B H EVANS, Fresno, Calif—I would like to refer briefly to the patient mentioned by Dr Paulson on whom Dr Samson and I operated almost a year ago

(Slide) This early film dated February, 1948, shows a bronchogram with occlusion of the right stem bronchus. This 14 year old boy at the age of 1 year was run over by a wagon. The accident was followed by a stormy course and hospitalization for three or four months.

(Slide) This is a film made approximately seven weeks after bronchial restoration of the right lung. This shows peripheral filling of the right lung, and I do not believe there is any significant bronchiectasis.

At the time of operation there was mucinous material filling almost the entire left lung, which had to be aspirated from the bronchial tree, the cultures from this secretion were entirely negative.

(Slide) This was made two months after operation. The right lung is well aerated, the diaphragm is elevated, is attached to the chest wall, and is movable on fluoroscopy.

(Slide) This is the most recent slide we have available. The right lung appears to be larger at this time. The trachea is now in the midline, and we hope that the lung will show further expansion.

DR WATTS R WEBB, Sanatorium, Miss—I want to emphasize the point that Dr Paulson has made, that these lungs that are atelectatic distal to a complete benign stricture do not need to be resected. Dr Evans and Dr Samson have demonstrated clinically that they can be re-expanded after a prolonged period of atelectasis. Dr Burford and I became interested in this some years ago and, in reviewing the pathologic slides of four lungs that had been resected at Barnes Hospital, after as long as twenty six years of total atelectasis, we found, surprisingly, that they looked almost normal except, of course, for the atelectasis. Experimentally we produced this in dogs by transecting the main stem bronchus, closing both ends, and leaving the lungs atelectatic for periods up to nine months. At the end of that time we reoperated, aspirated all the clear glairy mucus that has been described, and reanastomosed the bronchus. Each lung re-expanded promptly and completely. At a later date we were able to demonstrate a completely normal tracheo-bronchial tree throughout, we were able to demonstrate a perfectly normal vascular bed and also, by oxygen studies on the blood from both pulmonary veins to demonstrate that these lungs do oxygenate the blood that is flowing through them. These results were published in the June, 1953, issue of the *American Medical Association Archives of Surgery*, and we were able to predict with confidence that such would be feasible clinically, though we ourselves had not seen such a patient since the development of this concept.

I might make one suggestion on the case that has been presented by Dr Evans and Dr Samson, I think that this is not so much a case of incomplete re-expansion of the lung, as that, in the child, the lung did not grow as would the normal lung during the period while it was atelectatic.

DR J V THOMPSON, Indianapolis, Ind—I certainly appreciated the papers presented by Dr Abbott and Dr Paulson emphasizing the applications of these bronchoplastic procedures. My concern is with the application of these procedures to traumatic cases.

(Slide) This case was that of a 33 year old man who was admitted with a traumatic injury—a crushing injury to the right chest with multiple fractures of the ribs and a tension pneumothorax. The patient was treated at first by a closed thoracotomy with tube drainage. After twelve hours however, a rapid exudation of air occurred through the catheter, and his condition deteriorated. He was taken to the operating room and explored at once. There was found to be a complete severance of the right upper lobe bronchus and multiple lacerations of the right upper lobe necessitating lobectomy. Lacerations of the superior portion of the right lower lobe were repaired, and that lobe was saved. There was, in addition, however, a long longitudinal rupture of the trachea at the junction of the membranous and cartilaginous

urers, extending superiorly from the carina for 8 cm, then running transversely across the trachea and two cartilages. Oxygenation was maintained by a catheter in the left bronchus, and by use of the balloon on the catheter it was possible to keep the ruptured trachea closed until repair could be accomplished. This was done by approximating sutures after débridement. The patient was followed bronchoscopically, and there has been no evidence of stenosis.

The second case does not demonstrate an occluded bronchus for fifteen years as does the case of Dr. Samson, but the severed bronchus was present for two months before successful repair.

(Slide) The patient was admitted with a traumatic injury and was treated by a closed tube thoracotomy. The condition was complicated by severe cerebral injuries which necessitated waiting for eight weeks before a thoracotomy could be performed. A bronchoscopy was done shortly after admission and the left main bronchus was found to be occluded by a pulsating mass. Fortunately, the pleural space was filled by the atelectatic lung. At the time of exploration it was noted that the left main bronchus was completely severed. The ends of the bronchus were separated by the pulmonary artery. The ends were dissected free, trimmed, and reanastomosed.

(Slide) Good function of the lung was obtained, and no bronchial stenosis or other complication has occurred in the past year.

DR ROBERT FOX, Evanston, Ill.—I want to mention a traumatic case similar to that discussed. This one, unfortunately, was at a much higher level. A young boy was admitted to the hospital as an emergency following an automobile accident, with massive bleeding around the face and mouth, and obviously a compound fracture of the mandible. The resident noticed increasing respiratory difficulty, and the patient's neck, which was not lacerated, was starting to puff up and down with each respiration. After controlling the bleeding, the resident started to do a tracheotomy, made an incision, put a finger in to find the trachea, but it was not there. It had retracted about 5 cm down into the mediastinum. Fortunately, the boy had been kept face down to evacuate the blood from the pharynx. The resident was able to get a long tracheotomy tube in place while other resuscitative measures were done. The patient was then taken to the operating room and, with a few drops of local anesthesia, the incision was widened and the distal end of the trachea was brought up and found to be ruptured completely at the level of the second tracheal cartilage. There was also a vertical laceration in the membranous portion for about 1.5 cm downward. This was repaired and then a simple anastomosis was made of the tracheal stumps. Prior to this a new tracheotomy was made through about the fourth and fifth rings, a formal tracheostomy done, and the anastomosis completed. The recurrent laryngeal nerves were both completely ruptured, although the esophagus was not damaged. Of course, he had bilateral cord paralysis but at the present time he is back in school. He has a hoarse voice and some slight dyspnea on exertion.

(Slide) At this level there appears narrowing of the tracheal lumen. The lateral aminogram shows only slight narrowing. He does have a partial stricture and cannot take part in athletics, but he is able to handle himself well. We had not thought of doing anything about the stricture because of the bilateral cord paralysis. The tracheostomy was taken out about two weeks after the anastomosis.

DR DAVID H. WATERMAN, Knoxville, Tenn.—These two papers have emphasized that the preservation and restoration of function are certainly very fundamental concepts in pulmonary surgery. Probably the enthusiasm with which these concepts are embraced will vary directly with the conscientiousness of the surgeon. I would like to show two cases of bronchial anastomosis that may be instructive—our initial case and our most recent one.

(Slide) The first case is that of an 18-year-old man who presented himself with an emphysema on the right. Following drainage we noticed the atelectasis of the right upper lobe and demonstrated in this bronchogram complete block of the right upper lobe bronchus. Bronchoscopy revealed an adenoma completely blocking this bronchus and extending into the lumen of the main bronchus.

At operation, in July, 1950, we were reluctant to sacrifice the entire lung when we found that the adenoma had directly extended well into the main bronchus itself. The middle and lower lobes were anastomosed directly to the trachea after resecting the major portion of the right main bronchus with the right upper lobe. The next slide shows the final result several months later, with the right middle lobe and right lower lobe satisfactorily filling the right hemithorax.

(Slide) The next case is that of severe tuberculous stenosis in a nurse. The bronchogram demonstrates the marked stenosis of the left main bronchus, with complete block of the left upper lobe.

(Slide) Here is the postoperative film after resection of the destroyed left upper lobe and the stenotic main bronchus. The left lower lobe was anastomosed to the trachea.

I show this next slide to emphasize the importance of postoperative care. Three days after surgery the breath sounds were poor on the left, and obvious block is apparent on this x-ray. Bronchoscopy showed marked reduction of the lumen at the site of the anastomosis due to the temporary edema that always occurs. The final slide shows the complete aeration of the left lower lobe following relief of the temporary obstruction.

The recent article by Gravel in the JOURNAL brought out an important point, I think, in reporting the rupture of an anastomosed bronchus directly into the overlying artery, with fatal termination. We have seen similar rupture of a lobar bronchus into the artery.

To guard against this, we wrap a piece of pleura completely around the anastomosed bronchus in the same fashion as is illustrated in this slide showing our bronchial closure. This interposes an appreciable tissue barrier between the bronchus and the overlying artery, affording considerable protection, in our opinion.

DR CLIFFORD F STOREY, Portsmouth, Va.—I should like to compliment both Dr Abbott and Dr Paulson on their excellent presentations. My purpose in discussing Dr Paulson's paper is not to record my triumphs, but to present two cases, one of which developed an unexpected and distressing complication. I would like to ask Dr Paulson if he knows why this complication occurred and what could have been done to prevent it.

(Slide) This patient had a bronchial adenoma which proved to be of the carcinoid type. It involved the orifice to the middle lobe, the superior segment of the lower lobe, and the main right lower lobe bronchus. I believe that these diagrammatic sketches show the location of the lesion clearly and also explain adequately the type of operative procedure carried out. The lower lobe was resected as was the proximal portion of the middle lobe bronchus and the distal portion of the intermediate bronchus. The stump of the middle lobe bronchus was anastomosed to the stump of the bronchus intermedius just below the right upper lobe bronchial orifice.

(Slide) The film on the left is the preoperative one, the one on the right is a postoperative bronchogram. It is difficult if not impossible to see where the suture line is, and on postoperative bronchoscopy the lumen was entirely adequate.

(Slide) This patient was a comparatively young sailor who also had a bronchial adenoma of the cylindroma type somewhat larger than the previous one and situated in almost precisely the same position in the right lower lobe bronchus involving the margin of the superior segment and the orifice to the middle lobe, necessitating the same type of resection and anastomosis as that carried out in the previous patient.

(Slide) The preoperative chest roentgenogram is shown, he did not have complete bronchial occlusion in spite of the size of the tumor, and actually it is difficult to see in this film that he has a lesion. The x-ray on the right was taken approximately four months after resection and anastomosis, and I must say I was extremely pleased with the appearance of the x-ray. However, I made the mistake of carrying out a postoperative bronchoscopy and, much to my consternation, found that he had developed quite a stricture at the site of the anastomosis. He was kept in the hospital for several additional weeks and the stricture became progressively tighter so that it was necessary to go in and remove the middle lobe which we had attempted to save.

DR OTTO C BRANTIGAN, Baltimore, Md.—I wish to compliment Dr Abbott and Dr Paulson on the excellent work they have so beautifully presented. I would like to call attention to another type of graft which can be used to restore the trachea and bronchus a viable pedicle graft using the chest wall that is, the pleura, pericostum, intercostal muscles, intercostal artery, nerve and vein. I have found little chance to apply this type of graft since the original presentation by Dr Penton and me. We showed a successful reconstruction of the lower end of the trachea and both main bronchi. There have been two additional patients. One was a 30 year old woman with a bronchial adenoma involving the right main stem bronchus at the junction of the upper lobe. It was a question of either resecting the whole lung or doing some type of bronchial repair. The adenoma was locally resected, taking out about 3 cm of bronchus in length and about half of its circumference. The patient has remained well for two years.

The second patient was a 50-year-old man with a contracting localized carcinoma strangely enough arising from the upper lobe bronchus but contracting and invading the main bronchus, the lower end of the trachea, and the left main bronchus. We were able to resect the right lung, the lower end of the trachea and part of the left main bronchus reconstructing it with this particular type of viable pedicle graft. The patient did well for three days, and then began to have trouble. He died at the end of four days. The autopsy proved the justification for the operation, all carcinoma had been eradicated locally there was no lymph node involvement and no distant metastasis. The repair of the lower end of the trachea and left main bronchus was entirely intact and viable and certainly would have remained so. The patient died of a massive coronary occlusion.

DR DEWITT C DAUGHTRY, Miami, Fla.—I wish to congratulate the authors of the last two papers, for they have presented subjects which are of considerable interest and value. I should like to present four cases that fall into the same general category as presented in both these papers.

The first was a patient aged 54 with a history of wheezing and slight hemoptysis for one year. He had a negative x ray of the chest for the entire year and thus nothing further was recommended in the way of investigation or treatment. At bronchoscopy he was found to have a cylindroma almost completely obstructing the mid and lower trachea. Through a right posterolateral approach 7 cm of the lower trachea and posterior carina were excised. Small endotracheal tubes were placed in both stem bronchi and as the repair was being completed, this was replaced by a larger single plastic tube. The repair was accomplished by the use of tantalum mesh covered with fascia lata. A tube splint was left in the trachea much like the one presented in Dr Abbott's paper. This was removed after six days, and the patient's postoperative course seemed quite satisfactory until the eighth day. He developed a tension pneumothorax and a right empyema. An attempt was made to decompress the pneumothorax but was unsuccessful. The patient was in a rather debilitated condition and died with suppuration on the tenth postoperative day.

(Slide) This shows the specimen, 7 cm in length. The cut section is approximately 2 cm in thickness.

(Slide) This shows the tantalum mesh and fascia lata repair and reveals that most of the fascia lata has disappeared, thus explaining the tension pneumothorax which was impossible to cope with.

(Slide) Here is the larvnx, repaired trachea, and stem bronchi. One can readily see the extensive defect and that the posterior portion of the carina was resected.

Had the plastic tube splint been left in place longer, the outcome might have been different for it is not unlikely that the lung would have become adherent to the tantalum mesh thus sealing and making it airtight. The defect was so extensive in this individual that I doubt that a reinforced skin graft would have been successful. The use of intercostal bundles and the attached pericostum might have been a better choice in making the repair.

The three other cases were traumatic. One was a young Negro who had been involved in a motorcycle accident, sustaining a badly fractured larynx and upper trachea. The vocal cords and the adjacent mucosa had been almost completely detached. The larynx was rather badly fragmented and likewise the upper trachea was fractured and badly contused. The vocal cords and mucous membrane were reattached to the laryngeal cartilages and to the trachea. The fracture sites were débrided and closed in a rather meticulous manner. A tracheotomy was not performed, but the tray was kept available, and a resident was in almost constant attendance. A Levine tube was placed in the esophagus. The patient made an uneventful recovery including satisfactory restoration of his voice.

The next case was a Negro who had been involved in a battle of razors and had sustained a completely severed trachea just above the sternum. The thyroid gland had been severed and likewise the left internal jugular vein. The recurrent laryngeal nerves were intact. This patient's trachea was repaired with interrupted sutures without the use of a tracheotomy tube. This patient did not develop any subcutaneous emphysema and was discharged from the hospital on the sixth postoperative day, completely recovered.

The last case, and the most extensive injury of this type that we have seen, was a 52 year old man who was involved in an automobile accident. He was admitted to the hospital with a right tension pneumothorax, in profound shock, and exhibiting marked paradox of the upper anterior half of the right thorax. The skin over this area was intact but the ribs and muscle could not be identified. A closed thoracotomy was performed with the institution of strong suction. The tension pneumothorax remained despite strong suction for four or five hours. It was obvious at this time that the patient had a fracture either of the trachea or of a major bronchus. Through a right anterior thoracotomy approach, it was discovered that he had a fracture of the trachea just above the carina with only 1 cm. of the membranous portion still intact. The fracture extended across the right stem bronchus detaching the upper lobe bronchus. The azgos vein was severed and was actively bleeding into the tracheobronchial tree. The endotracheal catheter was guided into the lung and the left lung was kept inflated while the trachea and right stem bronchus were repaired with interrupted silk sutures. The patient was given the usual postoperative thoracotomy care and made a satisfactory recovery. Three months later he reappeared in the office with a considerable cough, and bronchoscopy revealed a considerable amount of granulation tissue at the site of the suture line in the right stem bronchus and, after removal of some of this granulation tissue, two or three sutures were visible, these were removed and the patient has had no further trouble.

This is certainly a very interesting group of cases. If proper treatment is instituted at an early date, the majority of these people may be salvaged.

DR. E. K. JOHNSON, Brooklyn, N. Y.—I have enjoyed the presentations and the discussion on the various topics. It has reshaped my interest in conserving lung tissue where possible by resecting the bronchial lesion and anastomosing the ends. I know that many of you have been impressed by the rather normal appearing pulmonary tissue beyond the lesion in the surgical specimen.

I would like to show some cases to illustrate this point. The first is that of a middle aged man who had a tumor of the right middle lobe. It was diagnosed as an adenoma. At operation it was found that the tumor extended into the right main bronchus, necessitating a bilobectomy, but a sleeve resection of the right main bronchus was done and the two ends anastomosed. On about the eighth day a bronchopleural fistula developed, but was controlled by drainage, and two months later a myoplastic flap occluded the fistula.

The second case was a 34 year old woman who had an adenoma of the right lower lobe bronchus. Since there was no evidence of suppuration, salvage of the middle lobe was done by resecting the right lower lobe bronchus above the orifice of the right middle lobe, and re-anastomosing it to the right main bronchus.

The third case I shall mention briefly. The patient had a large bulky tumor in the right upper lobe and, in attempting to resect the bronchus at the carina the opposite main bronchus was partly transected. The partly open end of the trachea was sutured to the cut end of the bronchus. Although I was greatly concerned I found that the closure was exceedingly easy and the patient had a most unremarkable recovery.

The first case was done two and a half years ago and a later examination of the specimen showed it to be a carcinoma. He is alive and well today. The second case was done 18 months ago, and the bronchogram shows that the anastomosis is secure and not strictured. The salvaged lobe aerates, does not trap air and seems to function well. The third case is too recent to comment upon. In each case bronchial secretions were retained for several weeks in the salvaged lobes and then they finally cleared.

I would like to ask the essayists if they believe that the salvaged lobe is functioning. If secretions are retained, would it suggest that air might be trapped later with emphysema and reduced ventilation? Is it a practical procedure? It appears to me to be practical but from their greater experience I should like to have their opinion.

DR GABRIEL P SELEY, New York N Y—I would like to comment on these two papers with particular reference to adenoma of the bronchus. At Mt Sinai Hospital we have had an experience with 82 cases over the past twenty years. Two of these were situated at the carina and certainly would be amenable to this type of therapy. However I would like to caution you on two points. In the first place many of these adenomas, if not all, have a marked iceberg type formation, that is, most of the lesion is outside the bronchial wall. Therefore with this type of operative procedure one will probably be able to remove this extrabronchial component with ease. Second in many cases a lymph node metastasis particularly by direct extension method is noted in these adenomas and in addition (and I do not think this has been stressed) there were four cases with distant metastases. Three of these patients have died of these metastases: two had had previous resections, one a lobectomy and one a pneumonectomy. In the fourth case there were just a few tiny nodules in the liver but this patient died of amyloidosis twenty six years after the onset of symptoms. Therefore for a permanent cure adequate resection of the extrabronchial component and the regional nodes is necessary for adenoma of the bronchus even though the malignancy is of very low grade.

DR LYMAN A BREWER III, Los Angeles Calif—These papers on tracheal and bronchial reconstruction are extremely important because they place increasing emphasis on preservation of lung tissue. Instead of excising lobes and lungs because of diseased tracheas or bronchi functioning portions of pulmonary tissue have been saved. The methods that have been so ably described this afternoon should be employed more extensively than is common practice today.

I wish to present briefly one technical point which we have found of great assistance in assuring the success of tracheal and bronchial grafts and anastomoses, that is the use of a pedicled pericardial fat graft which we presented last year at the San Francisco meeting of this Association. This graft will seal off the leaking of air in the anastomosis better than any other tissue that we have employed. It is a living graft which is adaptable both to bronchial and tracheal anastomosis and reconstruction.

In a case of tracheal resection performed from a left sided approach because we thought that it would be necessary to take out the left lung for a tracheal carcinoma the major portion of the trachea was resected making an anastomosis up in the neck and below to the carina and to the right main bronchus. Because of the difficult exposure of the right bronchus from the left chest we had trouble in effecting an airtight anastomosis. The closure however was made airtight with the use of a pedicled pericardial fat graft.

Our experience with pericardial fat grafts now totals over 300 cases. The grafts have remained viable for more than three years. As a matter of fact, the blood supply is so good that this method is being used by Dr. Arthur Vinclerg to provide additional blood supply to the myocardium. The use of the pedicled pericardial fat graft gives the operator

the assurance that the tracheal or bronchial anastomosis will hold *We have encountered no early leaks in the first three weeks after operation with this technique* The late leaks, from three weeks to one or more years, are less than 2 per cent

DR JOHN L POOL, New York, N Y—Dr Paulson has stated that pneumonectomy is the procedure of choice for carcinoma of the lung I am certainly in agreement with this statement This procedure, however, is not proper for those patients whose respiratory reserve is too low to survive without being a cripple after the total lung is removed or in cases with other physiologic needs for lesser resection I should like to call attention briefly to one danger of lesser resections This is a pathologic point, namely, that whereas the major lymphatic spread from bronchiogenic carcinoma is to the mediastinal lymphatics and also by the veins, one should not forget that there is another rich lymphatic channel in the submucosa of the bronchus and the trachea It is suggested that where resections of less than total pneumonectomy are contemplated, preliminary bronchoscopic biopsies of the tracheal wall and the major carina proximal to the level of contemplated segmental bronchial resection should be carried out In several instances after pneumonectomy we have had the misfortune to find at a later date recurrence within the tracheal wall Also unsuspectedly, on biopsy of the main carina in the study of patients in whom bronchiogenic carcinoma of the main bronchus is present and in whom pneumonectomy is contemplated, cancer has been found in those mucosal lymphatics

DR MICHEL LATARJET, Lyon, France—I appreciate the honor of being a guest of this Association I have been much interested in the contributions of Dr Abbott and Dr Paulson to this subject, and I would emphasize one point The bronchial reconstruction and almost end to end suture of the bronchi which has been traumatized by injury or by carcinoma requires, I think, a very sharp dissection All around the suture the peri-bronchial layers are cut off, that means bronchial arteries, some of the bronchial veins to the azygos, pulmonary nerves, and lymph ducts which are going in or coming from the pulmonary tissue I have been interested in the anatomy of the systemic blood supply to the lung, and I know it has been intensively studied by Dr Liebow and his associates, but it is still important to emphasize its value

I had the opportunity of performing such an operation on the bronchi three years ago in a boy, aged 13, after an accident The main bronchus of the right side was broken and was repaired by end to end suture Now I find that the lung is going very slowly but surely to a bronchiectatic status, and I am afraid the reason is that the bronchial artery was cut around the main bronchus On endoscopic examination the bronchus is free There is no stricture, no stenosis I can only say that when dissecting around the bronchi, one should be careful of the peripheral layers It is enough to have only 1 cm of this peribronchial layer, either on the anterior or the posterior aspect of the bronchi, to have a blood supply by the bronchial artery which preserves the lung tissue, which is placed beyond the anastomosis For there are numerous anastomoses between the bronchial arteries to the lobes, one of the branches of the arterial network, and one branch only, is able to supply the lung

DR OSLER ABBOTT, Emory University, Ga (Closing)—I think this film shows the supporting sutures, the extensive area of resection Here is a temporary endotracheal tube in place, and here is the prosthesis ready to slip in While that tube is being placed and adequately fixed, there will be a rather rapid interchange to slip the endotracheal tube within the prosthesis in the distal bronchus area This stresses the value of the many supporting sutures about the remaining strand of tracheal tissue Sutures anchoring the prosthesis into the upper end are placed, and after a little hyperventilation we are ready to step from the operating team endotracheal tube over to the anesthetist's tracheal tube which comes down through the larynx Here is the trade, and with that there is a minimal amount of anoxia It might be of interest to you to see the balloon expand within the prosthesis

I merely want to show the extent of resection of the adjacent trachea and bronchus involved in these radical right pneumonectomies, the value of the supporting sutures, and a method for transference of the one tube to the other for use. Now we present a brief demonstration of the size of the dermal graft which has been utilized up to a length of 10 cm. This flap of balsa wood we find helpful in keeping down strain on one's nerves and on one's instruments in placing the pins. A No. 24 silver wire suture is threaded through. It is very helpful to have one man anchor the pin toward which the other man is aiming as the wire is placed. Here the graft is being placed, and it will be sutured into place with multiple interrupted sutures. Over this will be placed a pedicle pleural graft, and particularly with the larger grafts is it mandatory to use the good vascular pedicle graft that Dr. Brewer described. We feel we owe him a great debt for that contribution.

DR. ROBERT R. SHAW, Dallas, Texas.—I want to thank all those who have discussed this paper. Dr. Abbott asked me to answer any questions that might come up with regard to his paper so that he might show this film. I cannot answer any questions with regard to the grafts he has used. We have used them, but we have not attempted any of the rather extensive resections such as he has shown here. Our main object has usually been to restore the airway by a bronchoplastic procedure rather than the use of a graft.

I would like to comment briefly upon the discussions of Dr. Fox and Dr. Daugherty. One of our residents, approximately three months ago, also encountered a situation where the trachea had been completely torn away from the larynx just below the cricoid cartilage. We have taught our residents and interns in the accident room to do an immediate tracheotomy on patients with crushed chests, especially if there is obvious interstitial emphysema. The intern, in doing the tracheotomy on the man who was just brought in, also could not find the trachea immediately and finally located it with his finger, grasped it with a clamp, pulled it up into the incision, and then called for help. The resident went down to the accident room and with no more anesthesia than that induced by the already consumed alcohol was able to do a very nice end-to-end anastomosis using fine wire, placing the tracheotomy lower down. This man fortunately had sustained no injury to the recurrent laryngeal nerves and had a good voice following the repair. I think that is important because in situations where there has been a traumatic tear or rupture of the trachea and tracheotomy has been used as an emergency measure without attempting to restore continuity immediately, plastic procedures that may be necessary to restore an airway and eliminate a tracheotomy can be very tedious and usually result in loss of voice.

INTRATHORACIC RUPTURE OF THE TRACHEA AND MAJOR BRONCHI DUE TO CRUSHING INJURY

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THE purpose of this paper is to report a case of rupture of the intrathoracic trachea complicated by avulsion of the right upper lobe bronchus with successful repair and to record a case of complete severance of the left main bronchus satisfactorily anastomosed two months after injury with restoration of pulmonary function.

REVIEW OF THE LITERATURE

A rather extensive review of the literature has failed to reveal a similar case of rupture of the intrathoracic trachea due to a crushing injury of the thorax which has been satisfactorily repaired. The case reported by Khelmskii¹ may have been similar but the paper was not available for review. Hanner and Cohen² successfully repaired a laceration of the intrathoracic trachea due to a penetrating injury, by the use of a full-thickness skin graft. Henry³ reported a case of traumatic partial rupture of the lower trachea which was discovered two weeks after injury when granulations obstructed the airway. The patient was treated by bronchoscopic removal of the obstructing tissue.

Zeuch⁴ in 1922 was able to collect 53 cases of rupture of the cervical subcutaneous trachea. Nine of these were treated by tracheotomy but none by open repair. Since then other cases have been reported in the literature.⁵ Scott described a complete laceration of the trachea and esophagus in the cervical region which was repaired by an end to end anastomosis with fine silk. Dysart⁶ patient had a severed trachea near the larynx. A tracheotomy was done and the airway was established by anastomosis over a tube. Nach and Rothman reported 7 cases in each of which there was a lacerated cervical trachea. All of these were repaired by direct suture. A tracheotomy also was performed on two of the patients. An avulsion of the cervical trachea was bridged by a plastic tube through which a tracheostomy tube was inserted by Longmire.⁷ Maisel and Dingwall⁸ were able to suture a lacerated cervical trachea and establish the airway. Knight⁹ treated a stenosis resulting from trauma of the cervical trachea by excision of the scar and replacement by a skin graft with the aid of an intratracheal mold. Fox¹⁰ recently mentioned the successful repair of an injury to the upper cervical trachea and larynx. Daugherty¹¹ satisfactorily repaired two such injuries of the subcutaneous trachea.

Kinsella and Johnsrud¹² in 1947 were able to find 37 cases of traumatic rupture of the bronchi in the literature. None of these were surgically repaired. Only one third of these patients apparently survived although no details were available as to subsequent complications. It is interesting to note that at least

two patients of this group were able to live over thirty years. These authors reported an additional case which was observed with an atelectatic lung and one case in which a pneumonectomy was performed two years after the injury. Priest¹⁵ described a case in which a thin membrane that was located in the bronchial tree, which resulted from injury, was opened through the bronchoscope to allow aeration of the lung. A similar case was noted in Kinsella's review. Anderson¹⁹ observed a patient with an atelectatic lung seven years after injury, but no surgery was carried out in this case. Santy, Bouchet, and Fraisse²⁰ reported a case of traumatic injury of the bronchus as did Giovanni²¹. Holinger, Zoss, and Johnston²² noted a complete occlusion of the bronchus two months after injury in a case in which there was no operative procedure. These authors also described two other patients with rupture of a bronchus. A pneumonectomy was performed upon one five weeks after injury and in the other, three years after injury. Hodes, Johnson, and Atkins²³ performed a pneumonectomy five months after a complete separation of the bronchus. Kirkpatrick²⁴ performed a pneumonectomy for a completely divided right bronchus immediately after injury.

Scannell²⁵ was able to repair the lacerated membranous portion of a right main bronchus with fine interrupted silk sutures. Two cases of rupture and two of occluded bronchi were described by Paulson²⁶ and Shaw²⁷ in which they utilized bronchoplastic procedures and the dermal graft. Daugherty¹⁶ was able to repair a rupture across the right main bronchus extending into the trachea, but the patient was troubled with granulations forming at the suture line as has been noted by other authors. Samson²⁸ and his associate recently described the case of a young man with a history of injury fifteen years prior to surgical reconstruction of the bronchus which resulted in an aerated lung.

Griffith²⁹ repaired a completely severed left main bronchus seven months after injury with an end-to-end anastomosis surrounded by a muscle graft. Weisel and Lake⁹ anastomosed a severed right main bronchus directly to the trachea forty-six days after injury.

Experimental work on the repair of the tracheobronchial tree has been carried out by several investigators. Grindlay and Clagett with their colleagues^{1, 32} demonstrated the technical difficulties encountered and the tendency to stenosis in using polyethylene tubing to bridge large defects. Craig, Holmes, and Shabart³³ likewise encountered stenosis in the use of a long polythene prosthesis. Bucher, Burnett, and Rosemond³⁴ utilized stainless steel mesh with some success in cervical tracheal defects but with poor results in the intrathoracic area. Daniel³⁵ repaired tracheal defects in animals with metal or glass tubes and demonstrated the difficulty of expectorating through prostheses and of maintaining them in place. Fascial grafts covered with surrounding connective tissue in his hands resulted in stenosis. Tiffel³⁶ pointed out the value of fascial grafts to cover windows in the tracheobronchial tree. Jackson and Tuttle with their colleagues³⁷ stated that fresh homologous tracheal transplants were of little value whereas short preserved homologous grafts were good and acted as bridges for new tissue growth. In their experience long grafts collapsed and

produced stenosis. They further pointed out that crushing of the bronchi caused poor healing of the anastomosis. Their best results were obtained from direct anastomosis after section of the bronchial tubes. Gelfoam was found by them to be a good bridge for small windows in the airways as was noted by Hanlon.³⁹ Mariangoni⁴⁰ found experimentally that homotransplants of lyophilized tracheal tissue were replaced in time with connective tissue which produced a satisfactory bridge for only short defects. Ferguson, Wild, and Wangenstein⁴¹ had poor experimental results with grafts but had good results with resection and end to end anastomoses, particularly where the mucosa was accurately approximated. Daniel and associates⁴² also had good success from direct anastomosis of the airways and found that results were equally good with the use of homologous or autogenous grafts to repair windows in the tracheobronchial tree. Davies, Edmiston, and McCorkle⁴³ found that, in animals, preserved homologous grafts produced a good repair of small windows and when utilized over acrylic tubes in large defects, but complete stenosis resulted from the use of these grafts alone in the replacement of large defects of the trachea. Carter and Strieder⁴⁴ have made an experimental study on the resection of the trachea and bronchi. Gebauer^{45, 46} has adequately shown the value of wire-reinforced dermal grafts for the repair of smaller defects and in the enlargement of stenosed areas in the tracheobronchial tree.

These methods and techniques have been applied clinically to repair defects in the major airways following radical excision for carcinoma or defects produced by the excision of benign neoplasms as well as for the surgical enlargement of fibrous stenoses of the bronchus.^{27, 45, 55} Penton and Brantigan⁵⁶ repaired a large defect in the trachea produced by the excision of a tumor by the ingenious method of utilizing a pedicled graft of the thoracic wall. The flap consisted of intercostal muscles, periosteum and pleura. This tissue was apparently selected in an effort to produce stability of the area by bony regeneration.

CASE REPORTS

CASE 1—The patient was a white man, 34 years of age, whose right thorax was crushed by a heavy concrete tile. The patient suffered from pain and dyspnea and expectorated bright blood. Cyanosis, tympany, absent breath sounds, subcutaneous emphysema, and bony crepitation were noted over the right thorax. There was a history of a previous gastroenterostomy. Roentgenologic examination disclosed a hydropneumothorax and multiple fractures of the upper five ribs on the right. The patient was given supportive therapy, and an intercostal catheter was inserted in the right pleural cavity for decompression purposes. Considerable relief was obtained by these measures. Early the next morning there was a rapid deterioration of the patient's condition. Severe hemoptysis and increasing dyspnea rapidly occurred. A continuous stream of air issued from the intercostal tube. A marked increase of the pneumothorax with displacement of the mediastinum was noted despite the decompression (Fig 2). A ruptured major airway was considered to be present.

A thoracotomy was immediately performed upon the right thorax. Prior to induction of anesthesia the blood pressure rose from 110/70 to 210/30 mm. Hg. Compression of the gas bag caused large quantities of gas to issue from the intercostal catheter which had been left in place as a safety factor until the thorax was opened, when air gushed from the incision. A large quantity of blood was evacuated from the pleural cavity. The right upper lobe bronchus was completely avulsed from the trachea and the lobe was so damaged by multiple lacerations that a lobectomy was performed. Oxygenation was maintained by

catheterizing the left bronchus with a long intratracheal tube. The tracheal stump of the bronchus was carefully trimmed of injured tissue and sutured with interrupted 00 silk. An extensive longitudinal rupture of the right tracheal wall was present at the juncture of the membranous and cartilaginous portions extending upward for about 8 cm from a point 1.5 cm above the bifurcation of the trachea and was not connected to the site of bronchial injury (Fig 1, A). The superior end of the defect also extended transversely and anteriorly across a fractured cartilage for about 1.5 cm. The azygos vein was divided and ligated. The edges of the torn tracheal walls were trimmed of devitalized tissue and the edges including the mucosa were carefully approximated with interrupted sutures of 00 silk. A flap of pleura was attached over the suture line with 00 catgut. The endotracheal tube was then retracted into the trachea from the left bronchus. Positive pressure was applied which demonstrated that the repair was airtight. The remaining lobes expanded to fill the hemithorax. A laceration of the superior division of the lower lobe was sutured with 00 catgut. Intrapleural protrusions of the fractured ribs which had lacerated the lung were rongeured away and the fractures were immobilized by sutures placed in the adjacent intercostal tissues. Three large catheters were inserted into the pleural cavity through stab wounds for decompression and the incision was closed. The patient's condition improved rapidly through the operative procedure.

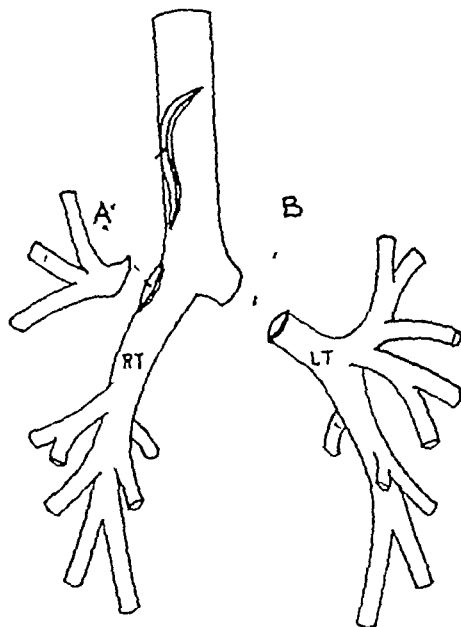


FIG 1—A Site of rupture of the intrathoracic trachea and avulsion of the right upper lobe bronchus in Case 1. B Site of severance of the left main bronchus in Case 2.

A soft tube was inserted through the nose into the trachea to separate the vocal cords in order to prevent the development of increased intratracheal pressure and to provide a means of aspirating pulmonary secretions. Periodic instillations of Pontocaine permitted the patient to tolerate the intubating tube. Oxygen was supplied through the tube until it was removed on the fifth day. The patient was able to swallow liquids despite its presence. Massive doses of antibiotics were administered and the blood loss was replaced by transfusion. The patient's condition rapidly improved during the postoperative period, and he was discharged on the fifteenth postoperative day with the remaining lobes well expanded (Fig 3).

The patient returned to his occupation and his condition was observed for a year after his injury. Tracheoscopic examinations were made at intervals of approximately three months, and there has been no evidence of stenosis.

This case is, therefore, that of a crushing injury to the chest resulting in rupture of the intrathoracic trachea which was successfully repaired with no development of stenosis. An additional complication of avulsion of the right upper lobe bronchus with severe lacerations of the lobe required lobectomy.

CASE 2—The patient was a white man, 58 years of age, who was pinned beneath an overturned tractor. He was in a semicomatose condition and a state of shock for which



Fig. 2—Roentgenogram illustrating the tension pneumothorax with mediastinal displacement despite decompression.



Fig. 3—Roentgenogram at time of discharge from the hospital following surgical repair of the injured intrathoracic trachea.

supportive therapy was administered. A left pneumothorax was decompressed by intercostal tube drainage. Tracheotomy was performed to maintain patency of the airway because of the presence of cerebral injuries, pulmonary edema and paradoxical motion of the thoracic wall due to multiple fractures of the ribs. Right hemiplegia and decubitus ulcers developed. The patient remained stuporous and semicomatose for a number of days. Neurosurgical consultation was had with Drs. E. Vernon Hahn and Paul Merrell. An electrocardiogram disclosed evidence of an old posterior coronary occlusion. An atelectatic left lung obliterated the space in the left pleural cavity by the third day, and a bronchoscopic examination revealed an obstruction of the left bronchus by lacerated bronchial tissues (Fig. 4). The nutrition was maintained through a Levine tube and by parenteral administration for the first week. Approximately a month after injury a bronchoscopy was again performed, which disclosed that the left main bronchus was obstructed by a membranous pulsatile mass of tissue. The diagnosis was that of a ruptured bronchus with resultant atelectasis of the left lung.



Fig. 4—Roentgenogram taken three days after injury demonstrating the atelectatic left lung, obliterating the hemithorax. The condition persisted for two months until surgical intervention.

Two months after injury the patient's condition improved sufficiently to permit a thoracotomy. Adhesions between the atelectatic lung and the thoracic wall were separated and a protruding fractured rib was resected away. An underlying laceration of the lung was repaired by suture. Dissection of the hilum disclosed a complete division of the left main bronchus with the pulmonary artery interposed between the separated ends of the bronchus (Fig. 1 P). The artery was mobilized by a tedious dissection through a mass of fibrous tissue and the bronchial stumps were exposed. The ends of the bronchus were obliterated by firm fibrous membranes. Both stumps were amputated to provide clean undamaged tissue for end-to-end anastomosis inasmuch as two cartilaginous rings on each side had been frac-

tured by the injury. A large quantity of thick tenacious seromucoid material was thoroughly aspirated from the distal stump and lung. A careful end to end anastomosis with good approximation of the mucosa was carried out with interrupted sutures of 00 silk, and a small piece of Gelfoam was used to surround the site of the anastomosis for reinforcement. The atelectatic lung became aerated readily after this procedure. The thoracic wall was closed following the insertion of two intercostal tubes into the pleural cavity for decompression.



Fig 5—Postoperative roentgenogram demonstrating the fully re-aerated left lung after anastomosis of the severed main bronchus

The postoperative course was marked by complete expansion of the lung. Secretions were aspirated through the tracheostomy tube which had been previously placed and was not removed until two weeks after the operation. Antibiotic therapy was maintained and the poor state of nutrition was improved by appropriate measures. A bronchoscopic examination, performed three weeks after the thoracotomy, revealed a widely patent left main bronchus with no tendency to stenosis or evidence of granulation tissue at the suture line. The patient was then discharged from the hospital.

The patient was subjected to bronchoscopic examination at intervals of three months for one year and no evidence of stenosis appeared at any time. Bronchograms disclosed no signs of bronchiectasis (Fig 5). The lung continued to function well but a residual hemiplegia has prevented the patient from resuming his previous occupation as a farmer.

The second case is thus that of a completely severed left main bronchus with interposition of the pulmonary artery successfully repaired by end-to-end anastomosis two months after crushing injury with restoration of pulmonary function uncomplicated by stenosis or bronchiectasis.

DISCUSSION

Rupture of the major intrathoracic airways, especially of the trachea, from crushing injuries of the thorax must be exceedingly rare as evidenced by the paucity of reports in the literature. Undoubtedly, there will be an increase in this type of injury with modern high-speed transportation. The mechanism of rupture must be related to a shearing force with a sudden increase in the intrathoracic pressure or to a sudden increase in the intratracheal pressure with the glottis tightly closed. On the other hand rupture of the cervical subcutaneous trachea was reported in 53 cases prior to 1922 and several additional cases have been reported since that date. The subcutaneous trachea also is more exposed to direct injury and the injury should be more readily diagnosed. Direct attempts to repair the trachea, however, were reported in only one-sixth of these cases.

Only 37 cases of ruptured bronchi were reported by 1947, and none of these were subjected to surgical intervention, with the result that two thirds of these patients died. An occasional patient lived a number of years with an atelectatic lung apparently due to spontaneous healing at the site of injury. The others probably suffered numerous suppurative complications. Twenty additional cases of rupture of the bronchus have been reviewed which were reported since 1947. Five of these were submitted to pneumonectomy and nine to plastic repair of the injured area. In one case a thin membrane formed at the site of injury and continuity of the airway was restored by bronchoscopic means.

If actual defects in the tracheobronchial tree should be created by injury fascial and dermal grafts appear to offer satisfactory means of replacing the smaller ones. Dermal grafts, however, seem to offer the best means at present for the repair of these small defects and for bronchoplastic procedures on stenosed major airways. Well-fitting autogenous and preserved homologous tracheobronchial grafts apparently may be utilized for the repair of small areas as they act as bridges for tissue replacement. These grafts, however, seem to be less practical for general usage. Various prostheses have not been too successful, particularly when utilized to bridge large defects because of their tendency to migration, interference with drainage, collapse and stenosis. Fascial grafts reinforced with tantalum or steel mesh probably have value for the replacement of large tracheobronchial defects.

Direct anastomosis and suture repair of the defects where feasible and the use of pedicled flaps offer the best means of reconstructing the tracheobronchial tree at present. Viable, smooth well approximating edges are essential to primary union and the prevention of granulations forming at the suture line. Maintenance of an adequate airway to the functioning respiratory area by intubation is essential during the operative procedure. A tracheostomy or an indwelling endotracheal tube during the early postoperative period is of value in the prevention of increased intrabronchial or intratracheal pressure with tension on the suture line and in the removing of secretions. An intratracheal tube may be maintained in place by the use of Pontocaine spray to prevent irritation or coughing. In those cases where surgical intervention was not carried out at

the time of injury and the patient survives, fibrous membranes cover the severed ends of the airways. The stumps, therefore, must be amputated and the sero-mucoid fluid trapped in the lung must be aspirated. The successful reconstruction of the tracheobronchial tree following crushing injury, as noted in the recent literature and as illustrated in the two cases just reported, leaves little place for pulmonary resection unless there is irrecoverable pulmonary damage.

The diagnosis of a ruptured major intrathoracic airway may be based on the presence of a tension pneumothorax with increasing dyspnea despite apparently adequate decompression and the almost continuous flow of air from the intercostal tube. The critical condition of the patient usually demands emergency repair to maintain oxygenation and relieve mediastinal compression. Spontaneous closure of a defect as noted previously may occur from occlusion by the adjacent tissues. In this instance operative intervention is indicated as soon as the patient's condition permits in an effort to restore pulmonary function and prevent suppuration of the lung in the future. Atelectasis occurring after thoracic injury, particularly with a history of pneumothorax or hemoptysis and bronchoscopic evidence of occlusion suggests the diagnosis in this circumstance. The plastic reconstruction of previously severed or occluded bronchial tubes apparently may be carried out several months to several years following an old injury with restoration of pulmonary function.

SUMMARY

The pertinent information contained in the literature has been reviewed and discussed. A case of successful repair of a ruptured intrathoracic trachea complicated by an avulsed lobar bronchus and a case of satisfactory delayed repair of a severed main bronchus resulting in a functioning lung following crushing injury to the thorax have been described in some detail. The apparent rarity of these injuries, the diagnosis, management and salient points in the reconstruction of the tracheobronchial tree have been discussed in the paper.

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OIL GRANULOMA AND LIPOID PNEUMONITIS A COMPLICATION OF OLEOTHORAX

REPORT OF SEVEN CASES

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THE use of mineral oil or paraffin as a method of plombage in the chest cavity has largely fallen into disuse in this country, although it has not been entirely discarded and it is still rather widely used in several foreign countries. Adams¹ only recently reported on the use of paraffin in over 200 cases, and stated that in none was there evidence of migration of the substance. Cutler² continues to use oil as a plomb in extrapleural pneumothorax and states that he has encountered no trouble with it. Gaensler and Strieder³ have recently used oil as plombage in a few selected cases and have observed no ill effects.

In contrast to these favorable reports, however, a survey of the literature will reveal that a number of cases have been reported in which oil granuloma formation or oil pneumonia has occurred. Matson,⁴ one of the earlier proponents of the use of oil, reported the formation of paraffinomata in 12 per cent of 50 cases in which oil in the form of Gomenol was used for collapse. Browning, Ray and Rotenberg⁵ reported in 1946 on the late investigation of 101 cases which they had studied six years earlier. Of the 75 patients which they had considered successful, eight had developed bronchopleural fistula and of these eight five had died. Oil aspiration presumably was a contributory factor. They found that after six years of oleothorax there was no case in which re-expansion of the lung occurred when the oil was withdrawn.

Of especial interest are those cases in which mediastinal oil granuloma has occurred with esophageal compression. We have been able to find eight such cases reported in the literature.⁶⁻¹⁰

Ballantyne, Clagett and McDonald⁶ in addition to reporting a case of esophageal obstruction due to oil granuloma also reported a case of oil granuloma in the chest wall and breast and another in which granuloma formation around the brachial plexus produced severe brachial plexus neuritis. They quote Livieratos as having reported cases with migration of oil into the chest wall and breast and to the opposite thorax.

Nagel¹¹ has also reported a case with contralateral migration and granuloma formation in the pleura and pericardium.

MATERIAL

There have been 76 cases at Nopeming Sanatorium in which mineral oil in the form of Gomenol has been used as a plombage method, either intrapleural or extrapleural. In addition there have been two cases treated by

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extrapleural plombage with paraffin. In seven cases complications have developed which were directly due to the presence of oil or paraffin and the granulomatous reaction which these agents produce. In addition to these seven cases, there were many others in which oil proved to be an ineffective therapeutic agent, and in a considerable number of cases complications due to tuberculosis occurred. However, of primary interest are those seven in which the oil per se caused the trouble.

CASE REPORTS

CASE 1—P V, a 68 year old former iron miner, was admitted to Nopeming Sanatorium Aug 19, 1940, with bilateral, far advanced tuberculosis and cavitation in the right upper lobe.

A right artificial pneumothorax was started on Aug 30, 1940, but was unsuccessful because of the development of tuberculous empyema. Repeated thoracenteses were done on the right but became progressively difficult, and multiple subcutaneous abscesses developed.

On May 25, 1941, the pneumothorax was converted to an oleothorax, which resulted in some improvement. However, in March, 1942, two pleurocutaneous fistulas developed, from which oozed only pus. These fistulas persisted in spite of many types of treatment.

In August, 1942, the patient had a cerebrovascular accident with a resultant hemiplegia, and his condition steadily declined until Oct 16, 1942, at which time he died.

Permission for autopsy was refused.

CASE 2—L M, a 22 year old male laborer, was first admitted in October, 1933, with moderately advanced pulmonary tuberculosis, bilateral, and with cavitation in the left lung. Anorectal tuberculosis was also present at this time.

In March, 1934, a fistula in ano was unroofed, and in July, 1936, a right pneumothorax was begun, followed by a left pneumothorax in September, 1936. These measures were ineffective and were abandoned. A right extrapleural pneumonolysis was done in March, 1942, and the resultant pocket was converted to an oleothorax. The same procedures were carried out on the left in March, 1944, resulting in marked improvement, and the patient left the sanatorium against advice in May, 1945.

In September, 1946, he was readmitted with a tuberculous empyema on the right side which was treated by aspirations. A subcutaneous oil abscess developed at the site of the aspirations. This was very difficult to treat. In order to obliterate this area, it ultimately became necessary to do a five rib thoracoplasty. Caseous necrotic material was found at the site of the abscess in the area of the interspaces. After thoracoplasty the patient improved and was discharged in 1948. To date he has remained well and is working.

CASE 3—M S, a 61 year old Polish housewife, was admitted to Nopeming Sanatorium, April 4, 1940, with far advanced bilateral pulmonary tuberculosis and cavitation in the right upper lung. She also had diabetes mellitus.

A right pneumothorax was begun in September, 1940, and was carried until December, 1941, when it was abandoned and oleothorax was substituted. This procedure was not effective in converting the patient's sputum but was carried for the next ten years.

In August, 1950, she began to expectorate oil, and immediately thereafter a new mottled type of infiltration showed up in the left lower lobe. The patient died three weeks later.

Autopsy revealed bilateral, active, pulmonary tuberculosis with a large, right sided cavity and a bronchopleural fistula. An empyema and oleothorax were encountered on the right, and an extensive pneumonitis was found in the left lung.

CASE 4—R R, a 35 year old housewife, was admitted to Nopeming in February, 1939, with moderately advanced, bilateral pulmonary tuberculosis and cavitation.

Pneumothorax was begun on the right within a few days after admission, and shortly thereafter an intrapleural pneumonolysis was done on that side. A left pneumothorax was

begun in April, 1939, followed by a left intrapleural pneumonolysis. Because of obliterative pleuritis the right pneumothorax was converted to an oleothorax on Aug. 23, 1943.

In August, 1946, a massive spread of disease in the right lung was apparent. A pneumoperitoneum was begun, but in November, 1946, the patient began expectorating oil, and a bronchopleural fistula became evident. The remainder of the oil was aspirated from the pleural space, but the patient went into acute right heart failure and died. Unfortunately, autopsy permission could not be obtained.

CASE 5—C. C., a 23 year old white girl, was admitted to Nopeming Sanatorium in December, 1938, with bilateral, moderately advanced pulmonary tuberculosis and bilateral cavitation. A left pneumothorax was begun in March, 1939, and shortly thereafter a left intrapleural pneumonolysis was done. Left pleural effusion which was treated with aspiration followed, a few months later it became an empyema. Oleothorax was substituted for the pneumothorax in November, 1941, and the patient was dismissed in 1942.

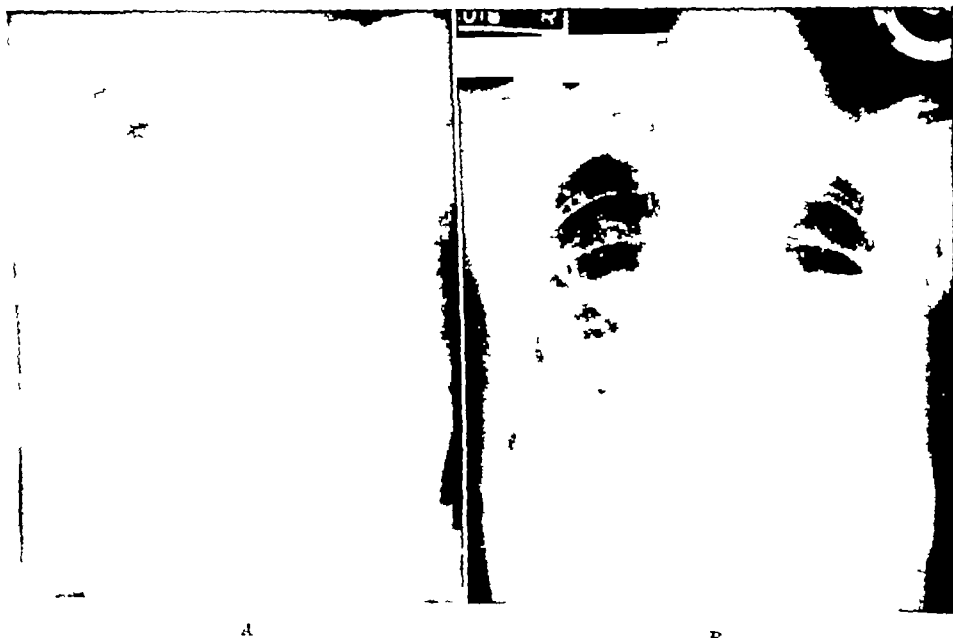


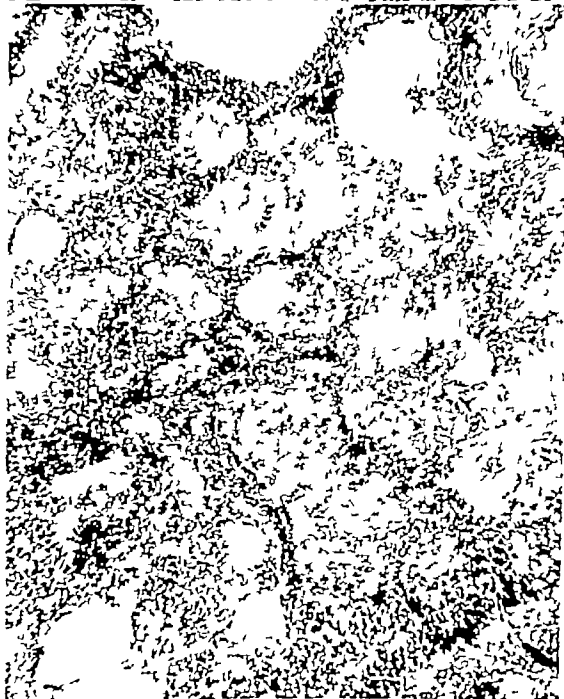
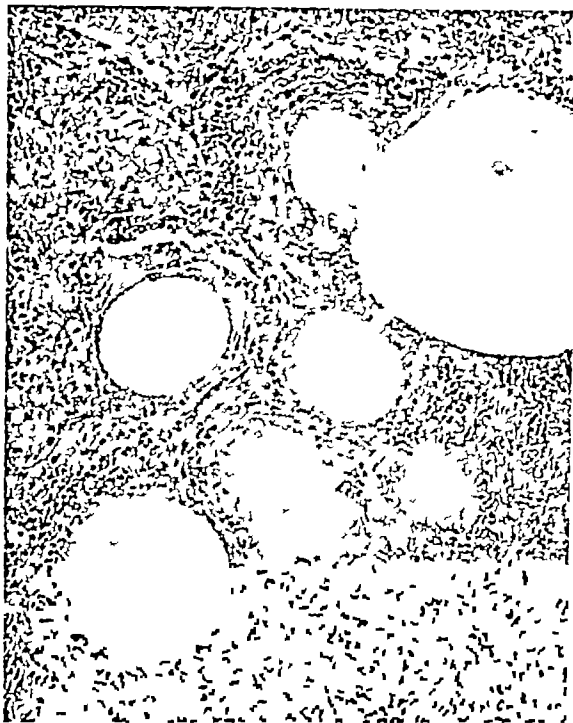
Fig. 1—Case 5. A, Roentgenogram made six months prior to development of bronchopleural fistula. B, Roentgenogram made three days subsequent to development of bronchopleural fistula.

She did well until January, 1950, when she again began to cough and to have fever. Later she began to cough up oil. Readmission films revealed an extensive pneumonia like process in the right lung (Fig. 1). The remaining oil was aspirated from the left chest, but the patient became markedly dyspneic and cyanotic and died March 18, 1950. Permission for autopsy was not obtained.

CASE 6—W. M., a 29 year old white laborer, was admitted to Nopeming Sanatorium in December 1934 with far advanced bilateral pulmonary tuberculosis.

A left sided pneumothorax was begun in January 1934, and a right sided pneumothorax was attempted but abandoned in October, 1937. The patient did well, however, and was discharged in June 1940.

In December 1943, he was readmitted with a left sided reactivation. A decision to continue the pneumothorax was made but tuberculous empyema soon ensued. The left pneumothorax was converted to oleothorax in September 1945.



B

Fig 2—Case 6. *A*, Oil pneumonitis showing spaces filled with oil and granulomatous reaction in the surrounding tissue. *B*, Oil pneumonitis showing an extensive exudate and numerous globules of oil filling the alveoli. These stained bright orange with Sudan IV fat stain.

While on a leave of absence from the sanatorium in August 1947 the patient developed a sudden coughing spell and coughed up profuse amounts of oil and became quite dyspneic and cyanotic. He was readmitted to the sanatorium as an emergency but died a few days later.

Autopsy revealed chronic, bilateral pulmonary tuberculosis with multiple cavities. A contributory cause of death was lipoid pneumonia throughout both lungs (Fig. 2). The diagnosis was proved by appropriate fat-staining methods.

CASE 7.—M. G., a 25 year old single woman, was first admitted to Hopeming Sanatorium Aug. 5, 1934 with moderately advanced, bilateral pulmonary tuberculosis. There was multiple cavitation on the right with a fibrocaseous appearing lesion on the left. Several attempts to begin a pneumothorax were unsuccessful.

On April 22, 1935, a right phrenic crush was done. Improvement was noted throughout the next two years, but in 1937 reactivation occurred. Because of this a left pneumothorax was begun. A left intrapleural pneumonolysis was done on Jan. 17, 1938, a right extrapleural pneumonolysis was done in March 1938, and a paraffin pack was inserted into the right extrapleural space the following June.

The patient convalesced well and returned to work, but in August, 1941 she suddenly experienced pain in the right upper chest and neck, and shortly thereafter a mass in the upper anterior chest wall was detected. This increased in size for a day and then became stationary.

On examination a hard mass 3 to 4 cm. in its greatest dimension was palpable in the second anterior interspace in the right midclavicular line. At this time it was decided that surgery should not be done but several months later when the mass had increased in size and there was more marked tenderness in addition to its being obvious that the muscles and breast were being infiltrated it was felt that removal of the pack was indicated.

On Feb. 14, 1942 the anticipated surgery was begun but only the extruded portion of paraffin was removed. However recurrence of the mass and symptoms necessitated reoperation in September 1942 with removal of the entire pack at that time.

The patient was last seen in 1946 but prior to that time she had progressed well.

DISCUSSION

Although there is no doubt that plombage of the lung with pulmonary tuberculosis is of occasional value in recent years methods have been developed which relegate such therapy to use in only a few well-selected cases. It seems therefore that the use of mineral oil or paraffin in the chest should be abandoned except for the rare case—as indeed it has been by most primarily because there are more effective ways of treating the majority of cases of pulmonary tuberculosis. In the second place the ever present danger of complications due to the foreign body reaction which the agent itself will often produce is a further argument in favor of the abandonment of the use of mineral oil and paraffin.

In our seven cases complications took the form of pleurocutaneous fistula, chest wall granuloma formation and oil pneumonitis due to aspiration following bronchopleural fistula formation, the incidence of such complications being approximately 10 per cent.

There seems to be no evidence that chemotherapy or better technique would have altered or will in the future alter the situation regarding the use of mineral oil in the treatment of tuberculosis except to outmode it further.

CONCLUSIONS

1 Mineral oil and paraffin, when introduced into the human tissues, act as foreign agents. This is true in the case of oleothorax, but the exact incidence of granuloma formation in such cases is unknown. In our experience the incidence is about 10 per cent.

2 Chemotherapy and newer surgical techniques make the use of mineral oil and paraffin in the treatment of tuberculosis a rare necessity.

3 Not only does the development of granuloma along with migration restrict the use of mineral oil, but as a form of treatment per se, in comparison with other methods of treatment of pulmonary tuberculosis, it is not of great value.

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EXPERIENCES WITH THE CYTOLOGIC EXAMINATION OF BRONCHIAL SWABBINGS IN THE DIAGNOSIS OF CANCER OF THE LUNG

A STUDY OF 602 CASES

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ACCURATE diagnosis of pulmonary cancer at the present time requires the combined efforts of the internist, the radiologist, the surgeon, and the pathologist. The greatest responsibility will always rest with the clinician who first studies the patient, suspicion of lung cancer and initiation of proper study are mandatory before any diagnostic team can function. Originally, the pathologist was limited to microscopic examination of small bits of tissue removed by bronchoscopic biopsy or expectorated by the patient. The pathologist's role in the diagnosis of carcinoma now is enhanced by the application of cytologic techniques, the significance of this method of tissue study is attested to by its widespread acceptance. The identification of cancer cells is a valuable aid to prompt diagnosis.

We have been interested in an uncomplicated technique that is well adapted to the cytologic study of fluids in general and that can be applied easily to the problem of diagnosis of carcinoma of the lung.

A method has been devised by one of us (J. B. H.) to study cells obtained by direct swabbing of the bronchial orifices at the time of diagnostic bronchoscopy. Examination of these simple wet stained suspensions approximates the results of more complex procedures. The conventional methods that employ rapidly fixed and stained films from sputa or bronchial washings can be exacting and time consuming. We have found that prompt examination of the wet stained suspensions obtained by direct bronchial swabbing is an accurate and economical method of cell study. Recently Jennings and Shaw² reported such a wet film method used in studying sputum, the interested reader can find also a summary of most of the studies of bronchial cytology to date.

It is the purpose of this presentation to describe the details of the method and to relate the experiences with its use in the study of material obtained from 602 patients each of whom was subjected to a single diagnostic bronchoscopy.

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MATERIAL AND METHODS

Technique —

Preparation of staining fluid The staining fluid used is similar to that previously employed for study of spinal fluid sediments³ and is made in the following manner

(National Aniline)

- | | |
|-------------------------------|-------|
| 1 Toluidine blue (histologic) | 1 Gm |
| Ethyl alcohol, 95% | 20 cc |
| Distilled water | 80 cc |
| Glacial acetic acid | 1 cc |
- Mortar the dry stain with the alcohol and slowly add the water Transfer the resulting solution to a glass, stoppered bottle and add the acid
- Mix 10 cc of the toluidine blue solution (1) with 10 cc of human blood serum
 - Centrifuge to remove the sediment The supernatant fluid is decanted and comprises the staining fluid It is kept in the refrigerator when not in use

Procedure at bronchoscopy A technician, specially trained in cytologic techniques, attends every diagnostic bronchoscopy A single tray has been devised (D B) which is brought to the endoscopic room and contains the basic material necessary for preparation of specimens (Fig 1)

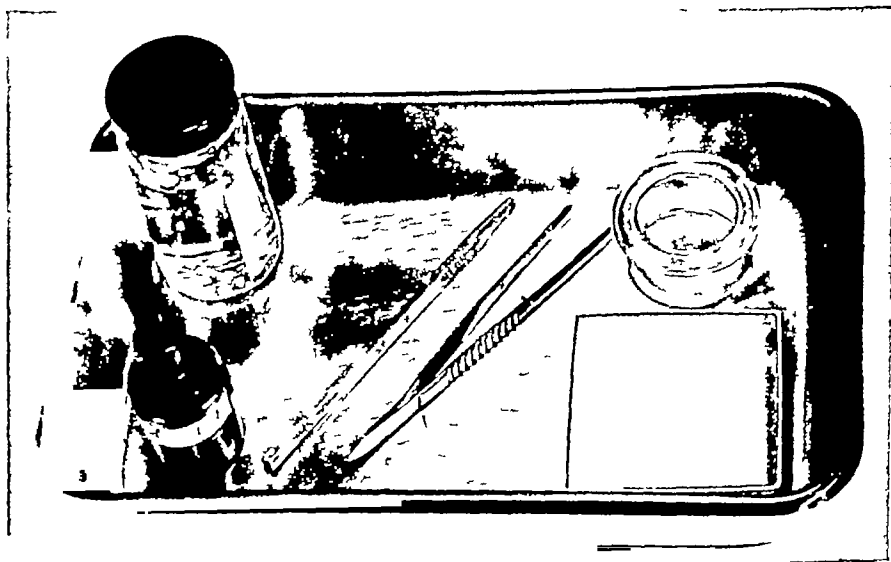


Fig 1—Simple tray containing glass slides forceps stain marking pencil cover glasses and acetic acid-alcohol fixative—all that is necessary for the technical part of the procedure.

Material is obtained through the bronchoscope by aspiration, by direct swabbing, or by biopsy Occasionally washings are made and the aspirated fluid is transferred to vessels and sealed for centrifugation and study of the sediment Small pledgets* attached to long applicators are used for swabbing

*Jackson sponge (Pilling Co)

the suspicious area within the trachea or bronchus. These pledgets, first moistened in saline, are rubbed over the area selected and are then passed directly to the technician for immediate preparation.

Separate specimens are smeared and air dried for bacteriologic study.

Preparation of suspension of bronchial swabbings

- 1 Place a large drop of staining fluid on a clean glass slide.
- 2 Remove the sponge from the bronchial sponge stick by grasping it tightly a few millimeters from the free margin with small forceps or snap. (This serves to isolate the surface covered with bronchial secretion from the unimportant yet absorbent portion of the sponge.)
- 3 Prepare the suspension by triturating the free margin of the sponge in a drop of staining fluid on a glass slide.
- 4 Apply a cover glass.
- 5 It is customary to prepare the material remaining on the sponge for examination by smearing it on a glass slide, fixing it immediately in acetic acid alcohol (95 per cent ethyl alcohol containing 3 per cent glacial acetic acid) and subsequently staining it by Papanicolaou's method.⁴ This fixing agent has proved stable after repeated usage and does not evaporate as does the commonly used ether alcohol solution. It is filtered before reuse. The rapidly fixed films are prepared last, the small amount of plasma absorbed by the sponge serves as an adhesive for remaining cells.

Usually two wet smears and two of the rapidly fixed type are prepared as described. The slides are then screened in the usual manner, utilizing the fresh preparations for immediate diagnosis. If no tumor cells are noted in the wet preparation the fixed slides also are screened. In no case has the stained fixed film been positive and wet film negative.

The method, at first glance, would seem to lack provision of material for later review. However, it is a simple matter to separate the coverslip from the slide and drop both into acetic acid alcohol fixative. The resulting fixed films subsequently can be stained as desired. The plasma-suspending medium serves as an excellent adhesive and the cells can be readily found after the slide and coverslip have been again mounted.

Histologic findings. The cytologic components of bronchial swabbings are similar to those seen in aspirations of bronchi. All types of bronchial epithelial cells are seen and readily identified (Fig. 2). Macrophages of varying stages of development are usually present. The histiocytes are sometimes difficult to interpret as is the case with other cytologic techniques. The other types of inflammatory cells are seen in varying numbers. Lymphocytes in groups are of particular importance as they occasionally can be confused with the cells of small celled carcinoma. The chromatin patterns and an irregularity of nuclear rims in the latter are valuable aids in differentiation.

The cytologic details that identify neoplastic elements in the wet suspension are the same as those that characterize these cells in the rapidly fixed films (Fig. 3). In the majority of cases tumor cells occur in clumps, a helpful feature for comparing aberrations in cell and nuclear size. Cellular hypertrophy alone is not considered diagnostic unless coupled with other findings. These changes include increased nuclear cytoplasmic ratio, anisocytosis, angulation of the nuclei and prominent nucleoli. Hyperchromatism is not a

prominent feature of neoplastic cells studied by this wet method, although irregular chromatin condensation is often apparent. Mitotic figures occasionally occur, but never are a prominent feature. A small celled neoplasm can be difficult to diagnose, as only a few of the previously mentioned features may occur—namely, a tendency to exfoliate cells in groups, chromatin condensation, and nuclear angulation. These features serve to differentiate them from groups of lymphocytes and basal cells.

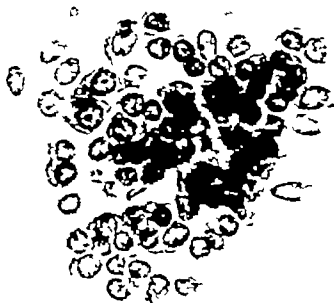


Fig 2—Clump of normal bronchial epithelial cells possessing all stages of maturation (Serum-toluidine blue stain $\times 400$)

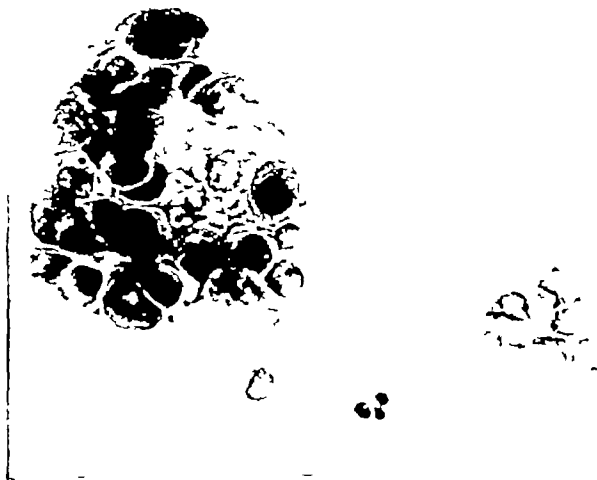


Fig 3—Neoplastic cells shed by a carcinoma of the bronchus. Note the hypertrophy of all elements, nuclear irregularity, and prominent nucleoli. A suggestion of pearl formation is present (Serum-toluidine blue stain $\times 400$)

The method does not always permit identification of the cytologic types of the individual neoplasms which exfoliate large cells. Occasionally, faint evidences of "pearl" formations or a waxy eosinophilia of the tumor cells suggest the diagnosis of squamous cell carcinoma but the red-to-orange colors of the Papancolaou technique are not apparent. Vacuolated cells are seen in some cases of adenocarcinoma.

A general warning concerning this method of study is necessary. To us, the changes associated with malignancy are intensified in this type of preparation as compared with rapidly fixed wet films. All cells, including normal epithelial flora and histiocytes, are larger than the equivalent cells in fixed smears, e.g., young macrophages are larger than those seen in fixed smears. Consequently the transition from the study of smears stained by the Papancolaou technique to this wet method must be made carefully to avoid an erroneous diagnosis of carcinoma.

RESULTS

Between early 1950 and Sept. 1, 1953 the bronchial swabbings from 602 patients were examined at the Cleveland Clinic. Of this group, 200 had clinically definite carcinoma of the lung. Diagnoses were histologically substantiated in 79 per cent (158 cases) of the 200 cases, either by bronchial biopsy, surgical exploration, or lymph node biopsy. The remainder (42 cases) were accepted clinically as hopeless cases of carcinoma of the lung. The cytologic findings were positive for neoplasm in 47 per cent of the 200 cases, and there were no false positive reports during this period. However, we do not believe that this figure represents the highest accuracy of the method. In comparison with this percentage of the entire series there was a 58 per cent evidence of positive results in the presence of neoplasm in the 400 most recent cases (1952 and 1953).

In contrast to the cytologic results bronchial biopsy was positive in only 26 per cent of the total group and was constant throughout the series. Table I gives a comparison and summary of the results that can be obtained by utilizing the cytologic technique described as an adjunct to bronchoscopy, the latter essentially consisting of a single examination in each case.

TABLE I. COMPARISON OF BRONCHOSCOPIC BIOPSY AND CYTOLOGIC DIAGNOSIS OF CARCINOMA OF THE LUNG (200 CASES)

	NUMBER OF CASES	PER CENT
Cytology positive	93	47
Cytology only positive	77	28
Bronchial biopsy positive	52	26
Bronchial biopsy only positive	14	7
Both positive	38	19

In addition to the cases diagnosed as positive the indeterminate diagnosis of the presence of suspicious cells was made in 23 of the 602 cases. Such a diagnosis is of some importance because 18 of the 23 cases were subsequently diagnosed as bronchogenic carcinoma. The remaining 5 patients suffered from some form of nonneoplastic chronic pulmonary disease.

COMMENT

It is apparent that information of definite value can be obtained by a simple method employed at the time of a single bronchoscopy. In this series no false positive diagnoses were made. The absolute reliability of a positive diagnosis is essential to the surgeon in those cases with the combination of a positive cytology, an indeterminate roentgenogram and clinically negative bronchoscopic findings.

Experience has led to an increased accuracy of the method and the results now approach those reported for the more difficult method of examining multiple sputa.⁵ The method, however, still lacks the sensitivity reported by Clerf and Herbut¹ in studying bronchial aspiration. In order to improve the sensitivity further, we believe multiple bronchoscopies would be necessary since the maximum effectiveness with a single specimen has been reached. Unfortunately, absolute accuracy is still not possible with any bronchoscopic and cytologic method, the responsibility for making the final evaluation of the case with negative cytologic findings rests with the clinician in charge of a patient.

SUMMARY

A simplified method of cytologic examination employing wet, fresh films prepared and studied as such from bronchial swabbings is described. The method is of no additional inconvenience to the patient and permits the pathologist to fix the preparation permanently for later or review examination.

Six hundred and two patients were so studied.

Of these, 200 were regarded clinically as having primary carcinoma of the lung, among these 158 (79 per cent) were histologically proved. The cytologic examination was positive in 93 (47 per cent) of this series of 200. An additional one third of the cases can be diagnosed positively prior to surgical intervention as compared with the results of bronchoscopic biopsy alone.

With a combination of bronchoscopic biopsy and cytologic study in the manner described, two thirds of the cases can be diagnosed positively, as evidenced by our results in studying the last 400 patients.

No false positive diagnoses were encountered in this series.

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THE MIDDLE LOBE SYNDROME AND ITS RELATIONSHIP TO CERTAIN ASPECTS OF MIDDLE LOBE DISEASE

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IN 1948 the term *middle lobe syndrome* was introduced by Graham, Burford and Maver,¹ to categorize a condition resulting from compression of the middle lobe bronchus by enlarged peribronchial lymph nodes with secondary changes in the distal parenchyma. It is now recognized clinically to be of rather frequent occurrence, and the term has gained wide usage. The exact definition of the middle lobe syndrome and the interpretation of its relationship to certain conditions affecting the middle lobe appear to be somewhat ambiguous. In order to ensure proper management a full understanding of this condition and certain primary diseases of the middle lobe simulating it is essential.

The particular vulnerability of the right middle lobe bronchus was first described by Brock^{2,4} on the anatomic basis of its length and narrowness, its acute angle of origin from the bronchus intermedius, and its close relationship to lymph nodes that drain the right lower lobe as well as the middle lobe and other lobes. He noted the frequency with which the enlarged hilar nodes of the primary tuberculous complex compressed and eroded the relatively soft middle lobe bronchus in infants and children. This concept of etiological association with tuberculosis soon became established and persists today. In 1946 Zdansky⁵ called attention to a nontuberculous etiology of the condition by presenting evidence of nonspecific lymphadenitis obstructing the middle lobe bronchus. Two years later, Graham, Burford, and Maver¹ described twelve cases of middle lobe bronchial compression by lymphadenopathy not caused by tuberculosis. References to the syndrome⁶⁻¹⁰ appeared with increased frequency following that report.

In our recent experiences with the condition treated surgically at Fitzsimons Army Hospital, we have encountered three cases caused by histoplasmosis and one by sarcoidosis. Such unusual etiology stimulated a perusal of literature pertaining to the middle lobe syndrome. After reviewing all cases in this hospital treated by middle lobectomy from 1946 to 1953, we were struck by the difficulty of defining an absolute classification of the syndrome so as to encompass the many variables encountered in each individual. Indeed, the complete picture can often be understood only when all of the varying factors are brought into proper perspective by the clinician, surgeon, and pathologist. The presenting symptomatology may be alike in most cases conveniently allowing classification as a separate syndrome whereas in truth the underly-

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ing pathologic agents affecting the middle lobe may be of considerable diversity. In fact, in the final analysis of the entire picture it may become apparent that a case originally diagnosed as middle lobe syndrome does not completely satisfy the classical criteria of bronchostenosis with perihilar lymphadenopathy demanded by strict definition and is, in reality, a type of middle lobe disease that closely resembles the syndrome. Five cases selected from this series are presented to illustrate certain features encountered in patients with the middle lobe syndrome and certain related diseases of the middle lobe.



Fig 1—Case 1. Lateral bronchogram showing contracted middle lobe with cylindrical and saccular bronchiectasis but no bronchostenosis.

CASE REPORTS

CASE 1—A 19-year-old white man was admitted to this hospital with a history of chronic productive cough, frequent colds, and occasional right lower anterolateral pleuritic pain as long as he could remember. He had had severe right-sided pneumonia when 5 years old and again when 13 years old. A chest x-ray shortly preceding this admission showed right middle lobe pneumonitis. On admission he was raising one-half cup of greenish-yellow mucopurulent sputum daily. Similar material was seen coming from the right middle lobe orifice bronchoscopically. Numerous smears and cultures were negative for *Mycobacteria*. A bronchogram demonstrated saccular bronchiectasis with crowding of the distal middle lobe bronchi (Fig 1). All other bronchi appeared normal. On Jan 18, 1949, thoracotomy revealed normal right upper and right lower lobes, but there were very dense adhesions and scarring about the right middle lobe hilus with bronchial thickening. No peribronchial lymphadenopathy was noted. Recovery, following right middle lobectomy, was uneventful.

Pathologic Examination—The lobe was moderately reduced in volume. No parenchymal defects were apparent grossly although thickened dilated bronchi were palpable. The middle lobe bronchus had been transected at its carina and gross mural thickening was evident at the origin of the medial and lateral segmental bronchi. No lymph nodes were seen, but microscopic sections disclosed residuals of lymphoid tissues closely applied to the major bronchial walls at their resection margins which, presumably, were contiguous with lymphoid tissue adherent to the proximal stump. These showed evidence of scarring and reactive hyperplasia consistent with chronic nonspecific inflammation. There was marked tubular and saccular bronchiectasis of classical variety particularly of the lateral segment. The lumina measured as much as 2.0 cm in circumference. Abundant evidence of active acute and chronic nonspecific bronchitis was obtained, one focus of mucosal ulceration was found deep within a branch of the middle segmental bronchus. Numerous foci of chronic bronchiolitis interstitial pneumonia and scarring were scattered throughout the pulmonary parenchyma. Cultures were negative for *Mycobacteria* and pathogenic organisms.

Comment—This case represents an example of bronchiectasis of the middle lobe that is frequently referred to as the middle lobe syndrome. Certainly most of the clinical features are suggestive of the syndrome but without bronchostenosis and peribronchial lymphadenopathy such a diagnosis is untenable in the strict sense. It could be postulated that the changes exhibited are only the result of insults from the childhood pneumonia. However, it also could be argued that the past pneumonic episodes resulted in acute peribronchial lymphadenitis. This perhaps with the edema of local bronchitis, produced temporary bronchostenosis and was predisposed to stasis secondary bronchitis and bronchiolitis within the lobe. Chronic interstitial pneumonitis became established which, with scarring contributed to the perpetuation of the bronchiectasis. The latter predisposed to recurrent bronchial infection long after the local hilar bronchial disease had subsided thus a vicious cycle was initiated. The peribronchial lymphoid tissue seen on microscopic section could represent the residuals of the previously involved lymph nodes. Despite the attractiveness of this hypothetical pathogenesis the available information necessitates a diagnosis of bronchiectasis of the middle lobe rather than a true middle lobe syndrome.

Case 2—A 75 year old white woman entered the hospital with a four to six month history of a hacking cough productive of one teaspoon of yellow sputum daily heavy ache in, over lower right anterior chest and a 10-pound weight loss associated with anorexia and increased fatigability. The past history was not contributory. A poorly circumscribed right hilar mass could be seen on x-ray in association with an atelectatic right middle lobe (Fig. 2). Repeated bronchoscopies revealed an indefinite mass in the right middle lobe bronchus. Biopsies were reported as chronic bronchitis and squamous cell metaplasia, but never definitely as neoplasm. Atypical cells were noted on Papanicolaou smear. Repeated cultures of sputum and bronchial washings were negative for tuberculosis and fungi. A tuberculin skin test was negative. Extensive antibiotic treatment had no effect. Exploratory thoracotomy was performed Dec. 8, 1952 with a preoperative diagnosis of probable carcinoma of the middle lobe. The middle lobe was found to be contracted and almost leathery in consistency. Several enlarged calcified lymph nodes were present in the right hilum and especially marked around the middle lobe bronchus. No tumor could be demonstrated, and a middle lobectomy was performed. For technical reasons the main middle lobe bronchus was divided distal to the densely adherent collar of enlarged peribronchial nodes. The patient was discharged improved after an essentially uneventful postoperative course.

Pathologic Examination—The lobe was markedly shrunken noncrepitant and fibrous. A few fibrous adhesions were present but the pleura was not remarkable otherwise. Resection had been complete but a few millimeter proximal to the principal carina of the middle lobe and no lymph nodes were adherent to the stump from which the nature of the lymphadenopathy described at surgery could be determined. The bronchi were uni-

formly dilated in tubular fashion and filled with thick, mucopurulent exudate. Extreme chronic nonspecific inflammatory reaction involved the bronchial mucosa, and there was marked transverse fibrous ridging. The parenchyma was diffusely atelectatic and had undergone considerable interstitial fibrosis. There was extensive bronchiolitis with active organizing interstitial pneumonia in the surrounding parenchyma. Cultures for pyogenic, acid fast, and mycotic organisms remained sterile.

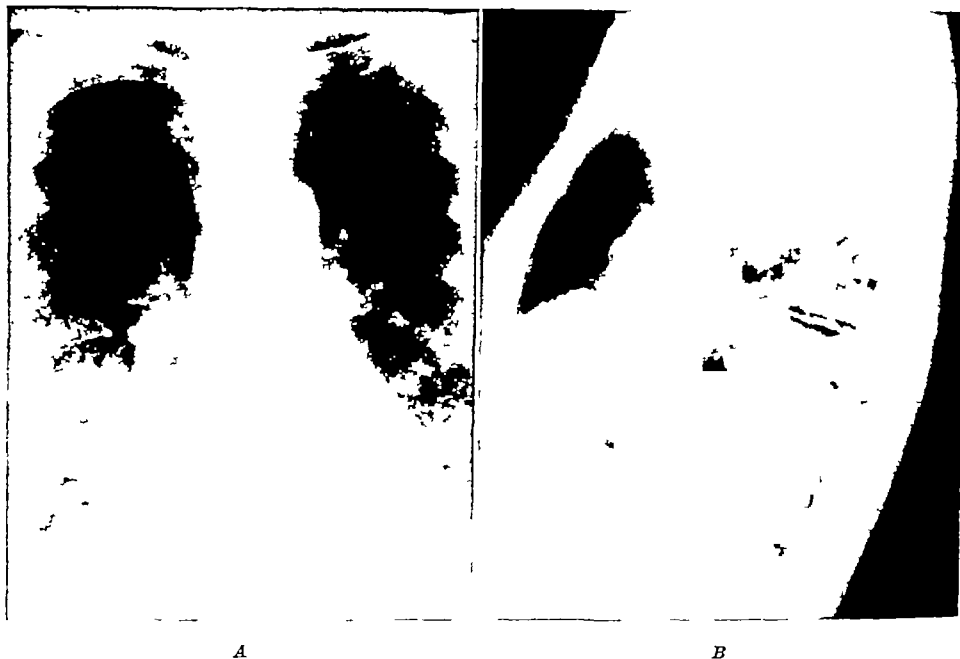


Fig 2—Case 2. A and B. Roentgenograms of atelectatic middle lobe. Note classical picture on lateral projection.

Comment—This case emphasizes the necessity for providing the pathologist with adequate material if etiological diagnosis is to be obtained. The surgeon elected to divide the middle lobe bronchus distal to the density-adherent collar of nodes and did not remove a node for examination. The finding of calcifications in the middle lobe hilar lymph nodes is strong presumptive evidence that a granulomatous disease existed, but whether this represents tuberculosis, histoplasmosis, coccidiomycosis or a more esoteric infection probably will never be known. The case, however, illustrates the extent to which the middle lobe is susceptible to extrinsic lymph node disease and the degree of secretory retention which may result from incomplete obstruction. The absence of sacculations suggests that the obstructive process was not of protracted duration and supports the possibility that active residual inflammatory disease persisted in the hilar lymph nodes. The extrinsic atelectasis must be ascribed to the retained secretions rather than directly to the partially stenosed stem bronchus. The short history in a patient of this age group in association with the other findings strongly suggests bronchogenic neoplasm. In all such cases, the question of malignancy finally must be settled by surgical exploration.

CASE 3—A 4-year-old white girl was admitted to the hospital in November, 1951. A twin brother had died of tuberculous meningitis in May, 1951. The patient's chest x-ray at that time was interpreted as showing right middle lobe atelectasis, probably nontuberculous. Since gastric washings were negative, the child remained at home on bed rest. The appearance of the chest was unchanged by x-ray over the next five months. A repeat gastric washing was reported tuberculosis positive on guinea pig inoculation in October,

1951 Streptomycin, 0.5 Gm every three days, and para aminosalicylic acid (PAS), 5 Gm daily, were started. The child was alert and active, but wheezing and occasional cough were noted. The chest was flattened anteriorly on the right with diminished breath sounds. Admission chest x rays showed hilar adenopathy, most marked on the right, with atelectasis of the right middle lobe (Fig 3, A). Repeated bronchoscopies were performed between November, 1951, and June, 1953. Marked distortion of the right bronchial tree with narrowing was noted. Initially, penetrating caseous lymph nodes were removed bronchoscopically to enlarge the lumen of the right main bronchus. The endobronchial changes slowly improved, but some irreversible damage persisted. Subsequently, a bronchogram revealed good filling of all segments with bronchiectasis of the right middle lobe and anterior segment of the right upper lobe. The hilar adenopathy had subsided by x ray prior to surgery (Fig 3, B). On June 19, 1953, the right middle lobe and anterior segment of the right upper lobe were resected. Both were atelectatic and fibrotic. Dissection of the atelectatic fibrotic diseased parenchyma was difficult because of the extensive peribronchial scarring. However, a careful search failed to reveal definite hilar adenopathy. An 8 mm primary complex was identified in the right lower lobe. The postoperative course was uneventful. Streptomycin and PAS were continued throughout the hospitalization for a total of nineteen months.



Fig 3—(A) Admission roentgenogram. Note hilar adenopathy and opacified middle lobe. (B) Roentgenogram shortly before surgery. The hilar adenopathy has regressed after prolonged streptomycin and para aminosalicylic treatment.

Pathologic Examination—The middle lobe was markedly shrunken and had no pleural lesions. The bronchi displayed marked tubular and saccular bronchiectasis with moderate trabeculation. That of the medial segment was significantly distended with mucus. All exhibited variable mucosal atrophy, scarring, and intense nonspecific inflammatory reaction. In addition to extensive chronic nonspecific bronchiolitis, the smaller air passages frequently were distended with retained mucus. Gross and microscopic examination of the parenchyma disclosed well developed atelectasis and focal areas of chronic interstitial pneumonia undergoing organization. No tuberculous lesions were encountered throughout and bacteriologic studies were negative for *Mycobacterium tuberculosis* fungi, and other pathogens. The only diagnosis permissible on pathologic examination was that of severe chronic nonspecific bronchiolitis.

Comment—The hilar lymphadenopathy associated with primary tuberculosis not uncommonly causes bronchial distortion and compression. In infants and children, in particular, caseous lymph nodes may progressively erode the bronchial wall to such an extent that endoscopic removal is necessary to maintain an adequate airway. Such a situation was present in this child. It was of interest to note that after nineteen months of continuous streptomycin and para aminosalicylic acid, no evidence of the caseous hilar adenopathy was noted on gross inspection at thoracotomy. In fact, to the pathologist receiving the resected pulmonary tissue, there were no absolute grounds upon which to base a diagnosis of tuberculosis. The subpleural focus identified by the surgeon in the right lower lobe, presumably the parenchymal component of the primary complex, might have served this purpose had it been removed for histologic examination. In the same way, lymphoid residuals occasionally have been demonstrated in such matted perihilar substance. The clinical information available, nevertheless, suggests tuberculosis as the probable etiological agent. This case illustrates the nonspecific destructive changes that may remain when the original disease that initiated the hilar lymphadenopathy is no longer apparent. Less severe cases that would have escaped surgical attention at this early stage may present themselves in later years as the middle lobe syndrome or nonspecific middle lobe inflammatory disease. It is not uncommon to see residual destruction in other lobes, such as was demonstrated in the anterior segment of the right upper lobe in this case.



FIG. 4—Case 4. *A* Roentgenogram with right hilar adenopathy due to sarcoidosis. *B* Bronchogram showing stenosis of right middle lobe bronchus secondary to lymphadenopathy.

CASE 4—A 26 year old white soldier was admitted to this hospital with a six year history of repeated colds and upper respiratory infections and a chronic cough productive at times of white and yellow sputum. He was hospitalized twice during the previous one and one half years because of pneumonia on the right side. Recently, the cough had become more productive, and blood tinged sputum had been noted. Bronchitis was thought to be the underlying cause of his respiratory complaints.

On admission he did not appear acutely ill or dyspneic. No cervical nodes were noted, but the spleen could be palpated two fingerbreadths beneath the costal margin. Skin tests for tuberculosis, coccidioidomycosis, and histoplasmosis were negative, all other laboratory tests were noncontributory. Chest x ray and fluoroscopy revealed increased prominence of the right hilum and a widened mediastinum (Fig 4, A). The tracheobronchial tree appeared normal on bronchoscopy. Bronchograms showed stenosis of the right middle lobe bronchus, a repeat bronchogram confirmed this obstruction in the right middle lobe bronchus (Fig 4, B). Palpation of the right lung at exploratory thoracotomy demonstrated fine diffuse infiltrations throughout, with many small nodules in the right middle lobe. Large boggy lymph nodes were present in the mediastinum. The extreme lymphadenopathy surrounding and compressing the right middle lobe bronchus made removal of this lobe technically difficult. The postoperative course following right middle lobectomy and mediastinal lymph node biopsy was uneventful.

Pathologic Examination—The lobe was of nearly normal size. It appeared well aerated except for an area of atelectasis within the lateral segment adjacent to the hilum. Numerous smooth 1 mm waxen nodules were palpable and visible within the anterior portion. The pleura appeared smooth and uninfamed. The lateral segmental bronchus and its immediate branches were stenosed by dense fibrous tissue throughout which were scattered numerous noncaseating naked tubercles consistent with sarcoid lesions. The remaining bronchi showed minimal tubular dilations and little or no inflammation other than scattered mucosal sarcoid lesions. The gross parenchymal nodules were identical histologically, containing numerous granulomas in perivascular and peribronchial pattern. Dense bands of fibrous tissue interconnected the lesions. The intervening alveolar parenchyma disclosed patchy atelectasis and emphysema. The several hilar lymph nodes which were received separately were enlarged to 1.5 cm but were relatively discrete and likewise contained numerous noncaseating tubercles. Asteroid and Schaumann bodies were encountered rarely within the Langhans giant cells which populated the granulomas in large number. Direct smears, appropriately stained sections, and cultures failed to disclose the presence of tubercle bacilli, fungi or protozoic organisms. A diagnosis of pulmonary and lymph node sarcoidosis was made without reservation.

Comment—This case is the first, to our knowledge in which sarcoidosis has been etiologically significant in the production of the middle lobe syndrome. Focal intralobar atelectasis is mentioned briefly by Longcope and Freeman¹¹ in their monumental discussion of this disease but no mention is made of compression of a major bronchus. This well may be due to the lack of perilymphadenitis and the relative absence of matting of lymph nodes considered characteristic. In this instance the obstruction is considered mild and probably intermittent as reflected by the relatively insignificant parenchymal alterations. That it was clinically important however is reflected by the recurrent pneumonitis. With the exception of the well developed scarring the pulmonary changes are considered to be reversible.

CASE 5—A 22 year old white soldier was admitted to this hospital because of five episodes of right middle lobe pneumonia during the previous two years that required hospitalization and extensive antibiotic treatment on each occasion. Each attack was characterized by malaise, chills, fever and right pleuritic pain with x ray evidence of right middle lobe pneumonitis. The past history otherwise was not remarkable. On admission he was afebrile and appeared to be healthy. Physical examination was essentially negative. Skin test were positive for histoplasmosis and negative for tuberculosis and coccidioidomycosis. Repeated cultures of sputum and bronchoscopic washings were negative for acid fast bacilli, fungi and tumor cells. X ray examination of the chest disclosed soft fluffy infiltrations in the right middle lobe suggesting pneumonitis (Fig 5). Bronchoscopy failed to reveal any abnormality, but by bronchogram the medial segment of the right middle lobe was noted to be irregular and bronchiectatic.

At thoracotomy Feb. 26, 1954 the right upper and lower lobes were normal but the middle lobe felt infiltrated. The major fissure was partially obliterated by adhesions

A collar of densely adherent lymph nodes presented about the main middle lobe bronchus. To divide this bronchus, it was necessary to transect an adherent grossly granulomatous node wedged in the junction with the bronchus intermedius. The postoperative course following middle lobectomy was uneventful.

Pathologic Examination—The middle lobe was moderately shrunken. The pleural surface was roughened by numerous fibrous adhesions presumably reflecting the numerous attacks of pneumonitis. The middle lobe bronchus had been severed 5 mm proximal to its primary carina, and adherent to the medial aspect of the stump were three small lymph nodes matted together into a single mass. They contained numerous discrete yellow granulomas from which caseous material could be expressed. Histologic study revealed these lesions to be of tuberculoid variety, surrounded by dense fibrous tissue. Epithelioid cells, plasma cells, lymphocytes, and Langhans giant cells were noted at their periphery, and their centers were composed of amorphous caseous debris. Moderate fibrosis of the surrounding parenchyma had occurred. Smears, cultures, and tissue sections were negative for tubercle bacilli, but sections stained by the periodic acid Schiff method contained small clusters of minute spheroidal organisms with a central punctum of chromatin and a thin capsule which were morphologically identical to *Histoplasma capsulatum* (Fig 6). These appeared somewhat distorted, swollen, and sometimes calcified, suggesting nonviability. Cultures failed to yield fungi.



Fig 5—Case 5. A and B. Roentgenograms showing nonspecific middle lobe pneumonitis secondary to histoplasmosis lymphadenopathy.

The lymph nodes, although not significantly adherent to it, produced visible compression of the medial segmental bronchus, the branches of which exhibited a moderate degree of tubular and saccular bronchiectasis and chronic nonspecific inflammation. Minimal chronic inflammatory and bronchiectatic changes were present in the lateral segment. The pulmonary parenchyma, particularly of the medial segment, was the site of mild patchy bronchiolitis and chronic nonspecific interstitial pneumonitis, a few of which were palpable. Some of these contained an alveolar exudate including cholesterol type macrophages consistent with obstructive etiology.

Comment—This case emphasizes the value of careful examination of obstructing lymph nodes in determining etiology and illustrates the necessity for more complete study of lesions bearing morphologic identity with tuberculosis, but in which acid fast organisms cannot be found. In two similar instances, histoplasma lymphadenitis and middle lobe disease, pathogenetically related to the middle lobe syndrome, have been seen by us. In one, a calcified node had ulcerated into the main middle lobe bronchus and presented to the surgeon as a broncholith when the bronchus was transected. In the other, extension of the infection had occurred in the region of the middle lobe hilus resulting in a broad focus of granulomatous pneumonia. It is felt that *Histoplasma capsulatum* is not generally appreciated as a cause of hilar lymphadenopathy capable of bronchial obstruction.



Fig. 1—Case 1. *Histoplasma capsulatum* in lymph node.

DISCUSSION

Fundamentally the middle lobe syndrome is the resultant of variable interplay between lymph nodes, bronchus, and distal lung parenchyma. The middle lobe bronchus is in the drainage pathway from the middle lobe and lower lobe parenchyma. The lymphadenopathy may be acute in nature and result in temporary mechanical bronchial compression which resolves more or less completely leaving no permanent parenchymal damage. Epituberculosis so called or an acute respiratory infection in a child may present such

a picture. If acute bronchitis occurs secondary to the lymphadenitis, inflammatory edema may further narrow the mechanically compressed middle lobe bronchus. If the infection is severe with peribronchial inflammation, permanent scarring and fibrosis of the peribronchial structures may result. This point is well demonstrated in Case 1. Severe peribronchial inflammation of this degree usually also involves the bronchial wall to such an extent that a poorly functioning or narrowed bronchus results, thus preparing the way for future infections. The acute caseous tuberculous nodes seen in childhood sometimes erode and penetrate the bronchial wall, resulting ultimately in changes in caliber in the bronchus as seen in Case 3. The more chronic calcified type of node seen in histoplasmosis as well as tuberculosis may also erode the wall, resulting in bleeding, further infection, or true broncholiths.

The nature of the pathologic changes in the distal bronchi and parenchyma depend upon such factors as the nature and duration of the obstruction and the presence or absence of secondary infection in the obstructed distal lung. If infection does not supervene, an atelectatic middle lobe may be an incidental finding on survey chest x-ray. As indicated above, the more common course is that of repeated bouts of infection leading to persistent lymphadenopathy, further bronchial compression, and increased obstruction resulting in a truly vicious pathologic and clinical cycle. Each repeated episode can only result in increasingly severe damage to parenchyma and bronchus, with ultimately the findings of gross bronchiectasis, chronic pneumonitis, fibrosis, and even lung abscess, bronchopleural fistula, and empyema.

The clinical diagnosis of middle lobe syndrome often means little in itself and can only lull the clinician into a false sense of security unless he is aware of the numerous etiological agents that may operate. The patient may present with cough, sputum, hemoptysis, and recurring pneumonitis, the classical symptoms of middle lobe syndrome, but the etiological agents responsible for the middle lobe abnormalities found clinically are variable. Although the pathogenetic mechanisms that produce the syndrome are fairly uniform, an understanding of the etiological agents is necessary in order to manage the case properly. In children, tuberculous hilar adenopathy is often responsible. MacPheison¹² lately has described a series of carefully studied cases of lung collapse associated with primary tuberculous lesions. Although carcinoma of the middle lobe is generally considered to be uncommon, Baldy¹³ has recently reported a series of cases. The possibility of an underlying neoplasm simulating the complex must always be considered in the older age group, and exhaustive studies may still leave the diagnosis in doubt necessitating exploratory thoracotomy. This is demonstrated in Case 2.

Valle¹⁴ has recently reported an unusual instance of middle lobe collapse due to hilar adenopathy related to inflammatory reaction secondary to lower esophageal trauma. Wohl, Israel, and Froheser¹⁵ have described a patient with middle lobe syndrome that may have been due to histoplasmosis. The diagnosis was suggested by a positive skin test but unfortunately could not be confirmed by additional careful studies. Our three cases of histoplasmosis were established by careful search of tissue sections stained by the periodic acid-Schiff method after routine staining failed to demonstrate the

presence of organisms Puckett¹⁷ has emphasized the usefulness of this procedure in demonstrating the tissue phase of histoplasma Exhaustive search for a fungus etiology when the surgeon provides adequate material undoubtedly allows specific diagnosis in many instances previously diagnosed as middle lobe syndrome due to granuloma of undetermined etiology

The middle lobe syndrome comes to mind when the clinician is confronted with a story of repeated respiratory infections in the middle lobe area productive cough, and hemoptysis A careful history and thorough laboratory studies including skin tests and cytology, may uncover the underlying etiology The chest x-ray may vary with the underlying pathology from the characteristic appearance of a completely atelectatic middle lobe to that of acute pneumonitis The right lateral projection may demonstrate changes easily missed on the anteroposterior film The presence of hilar lymphadenopathy, especially with calcification, should be carefully noted Bronchoscopy not only helps to evaluate stenosis and the origin of secretions or blood, but also permits a check for neoplasm foreign body perforating lymph nodes, and broncholiths A bronchogram often shows obstruction, in the main or segmental middle lobe bronchus beyond the vision of the bronchoscopist

The thoracic surgeon sees numerous cases diagnosed middle lobe syndrome that require thoracotomy to establish a definite etiological agent He is also fully cognizant of the many technical difficulties and potential hazards that may be associated with middle lobectomy in the presence of localized lymphadenopathy perihilar interlobar fibrosis, and inflammatory reaction The asymptomatic individual found to have a collapsed middle lobe by x-ray occasionally might be treated expectantly by a physician conversant with all aspects of the syndrome Similarly the need for surgical relief of symptoms in the nonspecific cases may be tempered accordingly by such factors as age, operative risk and the nature and severity of symptoms In general however, the treatment of this syndrome is essentially surgical and only rarely, when a benign etiology is established and the lesion proved to be stable, can the physician afford to temporize by careful observation

Because the term middle lobe syndrome is now well established and in wide usage it becomes necessary to define accurately its meaning and to understand completely its relationship to certain diseases of the middle lobe that closely simulate it Only in this way can existing ambiguity and confusion be dispelled and the proper management of patients so diagnosed be assured Originally the syndrome was defined to denote enlarged peribronchial lymph nodes compressing the middle lobe bronchus with certain changes in the distal parenchyma Without these limitations, imposed by definition the syndrome imperceptibly merges with a large number of other conditions affecting the middle lobe Therefore it is our contention that a diagnosis of middle lobe syndrome should not be made unless the following criteria have been met satisfactorily (1) hilar or peribronchial lymphadenopathy (2) bronchostenosis and (3) the variable changes in the distal parenchyma, previously mentioned It becomes apparent that the final interpretation may be possible only after reviewing the complete clinical surgical and pathologic findings

Lymphadenopathy may have been demonstrated during the acute phase by x-ray or bronchoscopy and yet not be apparent at a somewhat later stage on gross or microscopic examination. In the same way, bronchoscopy and/or bronchography may have shown middle lobe bronchostenosis that proves to be reversible when the acute phase of pneumonitis, lymphadenitis, and bronchitis subsides. In these somewhat borderline examples of the syndrome, one can appreciate the need for a complete evaluation of *all* data without which the condition could be variably interpreted by internist, surgeon, and pathologist.

These alterations initiated by peribronchial lymphadenopathy may involve other lobes and segments independently or concurrently with the middle lobe. The middle lobe, of course, is most commonly involved, but when, in addition, several segments or lobes are affected, we believe the condition no longer fits the true definition of middle lobe syndrome.

As previously mentioned, the term middle lobe syndrome has become established by usage and will probably persist. Rather than belabor the need for this questionable separate designation, we feel that the problem can be met best by accurately defining the syndrome and correctly interpreting it and its relation to certain other middle lobe conditions. In this way the term will take on a more definite meaning and form a more concrete basis upon which to establish definitive treatment.

SUMMARY

The term middle lobe syndrome is defined and its relationship to certain diseases of the middle lobe that may simulate it discussed. The wide use of the term demands a clarification of the existing ambiguity of definition and interpretation so as to ensure proper management of the individual so diagnosed. The syndrome must meet the following criteria in reference to the middle lobe: (1) hilar or peribronchial lymphadenopathy, (2) bronchostenosis, and (3) certain changes in the distal parenchyma discussed in the body of the paper. The final interpretation is often possible only after the complete clinical, surgical, and pathologic data are reviewed.

Special stress is laid upon the diversified etiology that may be present in contrast to a more constant symptomatology. The concept so strongly held in the past, of commonly associating the syndrome with tuberculosis must be abandoned. Selected case reports of middle lobe syndrome and closely related middle lobe disease are included to form a basis for discussion of certain aspects of the problem. Among these are examples due to sarcoidosis, histoplasmosis, tuberculosis, and nonspecific etiology.

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LEIOMYOMA OF THE ESOPHAGUS

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THE first report of an esophageal tumor presumed to originate from muscle was made by Monro in (1797),⁵⁰ however, he failed to furnish histologic evidence thereof. Such was also the case in the similar reports of Bell in 1816,⁴ Lippich in 1838, Meyer 1882,⁵³ and Kraus in 1902.

In 1863, Virchow⁵³ first recorded the anatomopathological characteristics of a leiomyoma of the esophagus. Numerous other case reports followed, but were almost all *post-mortem findings*.

In 1932, Bezza⁶ reported five personal cases and reviewed thirty-nine from the literature. He concluded that esophageal myomas were very rare and of little practical importance.

In the last twenty years there has been a progressive increase in the recognition of these tumors. In this period the reports were, for the most part, made not by anatomopathologists, but by surgeons. Ohsawa,⁶¹ Brock,⁹ Harrington and Moersch,³⁸ Calmenson and Clagett,¹³ Schafer and Kittle,⁷⁴ Kenworthy and Welch,⁴² Harrington,³⁹ Placitelli,⁶⁶ Hurwitz,⁴⁰ Manzocchi,⁵² Lortat-Jacob,^{45, 47} Chi and Adams,¹⁶ Cornell and co-workers,¹⁹ Daniel and Williams,²¹ Dugan and Meagher,²³ Rossello,⁷¹ Stojanovic and Jelijevic, Goldman and Masters,³² Garlock and Stegmaier,³⁰ and Chalnot and co-workers¹⁵

The reports in the literature on leiomyoma of the esophagus can be divided into three periods: an early one, from Monro (1797),⁵⁰ to Amtschkow (1911),³ which includes findings of a principally morphologic character, a second period up to Bezza (1932),⁶ when investigations were chiefly directed to histogenesis, and a third or contemporary period since Ohsawa's first operation (1933),⁶¹ during which surgeons have treated the problem from a diagnostic and therapeutic point of view.

Frequency—It seems probable that the frequency of these tumors is not so limited as is generally believed, particularly since certain numbers of cases escape diagnostic confirmation, owing to their lack of symptoms.*

The general incidence of leiomyoma in the alimentary canal, which was established by Smith in 1937, Golden and Stout in 1941, and Oberhelman and co-workers in 1952,⁶⁰ puts the greatest number of cases at the level of the stomach and the small intestine. The lowest percentage is met with, according to Smith, at the level of the colon, while in the opinion of Golden and Stout, and Oberhelman and co-workers, it is encountered at the level of the esophagus (Table I).

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In a very recent publication (J. THORACIC SURG. 27: 1, 1954) Richard H. Sweet reports on 20 cases of benign tumors of the esophagus of which 17 were classified as leiomyoma.

TABLE I. SUMMARIES OF TOTAL REPORTED CASES OF LEIOMYOMA

AUTHOR	YEAR	TOTAL NO CASES REPORTED	LOCATION			
			ESOPHAGUS	STOMACH	SMALL INTESTINE	COLON
Smith	1937	513	50	321	109	33
Golden and Stout	1941	975	51	629	197	98
Oberhelman, Condon and Guzauskas	1951	1105	66	705	225	109

In regard to the relative frequency of leiomyoma of the esophagus it has not been possible to establish an exact and complete comparison either with dysphagias in general or with the other benign and malignant neoplastic forms of the esophagus. Such a comparison has been made by only a few authors and most of the cases in the literature have been reported as isolated instances, without any reference to the absolute or relative frequency of the tumors. Of 4 000 patients with dysphagia Vinson, Moore, and Boromv of the Mayo Clinic, found three benign esophageal tumors while Moersch and Harrington reported 15 among 11 000 cases of dysphagia. These last two authors, surveying a series of 7 459 autopsies at the Mayo Clinic, noted 44 benign tumors of the esophagus of which 32 were leiomyomas. Chi and Adams have reported a list of all the cases of benign esophageal tumors published up to 1948 which showed a 50 per cent greater frequency of esophageal myomas over all other benign neoplasms in the same location. The figure quoted by Calmenson and Clagett (70 per cent) is even higher.

Regarding the frequency of the benign tumors of the esophagus in respect to carcinoma, Chi and Adams reported that in the Clinics of the University of Chicago only two benign tumors of the esophagus were noted as compared with 246 carcinomas in the same location with an incidence therefore of less than 1 per cent in comparison with the malignant tumors.

SEX.—The sex distribution of the cases in the literature is as follows:

Males	52
Females	26
Sex not identified	22

Bezza attributes such greater incidence to the more frequent causes of irritation to which the esophagus in the male sex is subjected (dietetic disorders, alcoholism, etc.). This hypothesis would seem more applicable to tumors which have their origin in the mucosa rather than to those which arise from the muscle.

AGE.—Regarding frequency in relation to age the percentages are as follows:

From 11 to 20 years	Cases
21 30	5
31 40	8
41 50	9
51 60	21
61 70	15
71 80	13
Age not determined	7
	22

From these statistics the deduction might be drawn that leiomyoma of the esophagus is more frequent after the age of 40, as is maintained by some authors,^{6 13} who base their deductions on analogous statistical, albeit numerically inferior, conclusions.

Such conclusions do not seem to be acceptable when dealing with benign tumors of slow development, which are for the most part asymptomatic, at least in the early stages of the disease.

It may be added that the ever increasing frequency of the neoplasms noted in young patients, possibly thanks to improved diagnostic technique, has drawn some authors, Rossello, for example, to conclude that it is the young who are chiefly subject to such afflictions.

Location—The cases in the literature are distributed thus:

Leiomyoma of the upper third	14
Leiomyoma of the middle third	23
Leiomyoma of the lower third	37

For gastroesophageal leiomyomas Manzocchi²² has correctly advocated separate classification, including in such a category the cardiac region extending to the gastric wall.

PATHOLOGNETIC CONSIDERATIONS

The histogenetic problem of these tumors is continually under discussion but there are three principal theories. The most accredited of these^{3 20} places the origin of the tumor in the esophageal muscle membrane, while Aschoff also suggests that it may originate from the muscularis mucosa, a theory which is, however, excluded by Fischer.²⁰ To admit such a contingency, according to Rodino' di Miglione,⁶⁰ a demonstration of continued affinity between the muscle tissue of the esophagus and that of the tumor is not necessary, however, indispensable serial sections of the entire mass would be. The transition zone can be very circumscribed and limited to one determined sector only.

The second theory, which puts the origin of esophageal leiomyoma in the vessel musculature, has been discussed by many authors, but almost unanimously rejected.^{26 82} Some authors, such as Busineo, hold it to be possible, thereby attributing a very rare origin to the esophageal area, especially for those tumors in which vascularization appears to be autonomous and well ordered. Rodino' di Miglione also considers the hypothesis of vascular origin rather forced for such an organ as the esophagus, in which an abundance of muscle tissue already exists. It might be accepted to explain the rise of leiomyomas in organs in which smooth muscle fibers do not normally exist.

The third theory, according to the well-known ideas of Durante and Cohnheim, considers the origin of esophageal muscle tumors, as of analogous neoplasms of other parts of the alimentary canal, to be in aberrant nodes of embryonic muscle tissue. Di Falco²² accepts this hypothesis on the basis of a personal finding of a mediastinal myomatous node, independent of the esophagus and adhering to a lamina of tissue structurally similar to the esophageal wall.

This theory would not oppose the two preceding ones since the aberrant germ can originate from various layers of the esophageal muscle or from the muscle fibers of the vessels.

PATHOLOGIC ANATOMY

Macroscopic Appearance—This often varies, depending on how and when the tumor is discovered. In asymptomatic cases, and in incidental post-mortem findings, the tumor, rounded or oval in shape, has a limited size, rarely larger than a hen's egg.^{6 18 24 37 82} In those cases which have been clinically confirmed, the neoplasm generally presents a more varied appearance, plumobular in shape like a horseshoe, or a plaque, generally of larger size.

The tumor is firm in consistency, except in a limited number of cases,^{27 82} the outer surface is smooth or rounded. A cross section shows, in almost every case, gray white, mother-of-pearl color. Sometimes reddish striations are visible, due to better vascularization, or dark brown areas of tissue degeneration,⁵⁰ or hard zones of calcification.^{3 6 41}

According to whether the point of origin corresponds to the muscularis mucosae or is intramucosal, such neoformations can arise in the esophageal lumen and remain intraluminal, or develop in the periesophageal area.

If it advances toward the esophageal lumen, the tumor is completely or largely covered by mucosa, by the adventitia if it develops toward the outside. Frequently, placed like a capsule, it appears surrounded by connective tissue, which permits discrete mobility. In other cases, it seems to be part and parcel of the esophageal muscle, characterized by a certain immobility, while the mucosa and adventitia remain free and mobile on its surface.

Antschikow's opinion, according to which only old or very large tumors are capsuliform, is in contrast to the findings of small, slow-growing and distinctly encapsulated leiomyomas of the esophagus, as maintained by Tschlenow, Businco, Rodino, di Mighione, and Garlick and Stegmaier.

The esophageal area above the tumor mass sometimes presents dilation of the lumen and hypertrophy of the muscle, with appearances similar to the widespread myomatosis⁷ described by Lortat-Jacob.⁶⁰

Classification—Almost all authors report on benign tumors of the esophagus without following any classification. Some^{30 38 63 71} distinguish two categories: tumors originating from the mucosa and submucosa, including fibromas, adenomas and fibroadenomas; and tumors which arise from the rest of the esophageal wall, called by Garlick and Stegmaier extramucosal, the most frequent of which are leiomyomas.

A characteristic of the first group would be pedunculization in the lumen, and of the second intramural location. This affirmation, however, does not always correspond to reality, owing to the existence of leiomyomas possessing a long pedicle and arising in the esophagus.^{15 57 61}

As far as concerns esophageal leiomyomas, it does not appear to me that an exclusive classification was made in the past in their case.

Recently Rossello has advocated the distribution of cases in the literature into one of the three following groups:

1. *Leiomyomas with isolated nodes*. This type generally appears as a single node varying in size from a grain of rice to a walnut and is usually located at the top of the trichial bifurcation or at the cardia. The smallest myomatous nodes observed have been at the cardia.^{44 61} A leiomyoma in isolated form can even though very rarely appear at multiple sites.^{11 37 52}

2 *Leiomyomas with multiple nodes* These generally reach the dimensions of an egg, although they are sometimes larger than a fist. Limited to a tract of the esophagus which generally represents the greatest axis of the mass, the tumor appears as if made of multiple myomatous nodes crammed one on top of the other, situated equally in the mid third or lower third of the esophagus. The dimensions of the single nodes generally vary in size from a hazelnut to an almond.

3 *Leiomyomatosis or widespread myomatosis* In an area of the esophagus, variable in length, but generally from 5-12 cm., numerous myomatous nodules are to be found in one or more of the muscle layers. These are evident after bisection of the esophageal wall, which itself shows only a greater firmness than usual to palpation.

Sometimes true nodular formations do not exist, mere myomatous zones being included in the thickness of the muscle that are recognizable to careful palpation as a granular and diffuse thickening of the wall, and visible only under the microscope (the external appearance of the esophagus is hardly or not at all modified, and can be said to be normal). To this last category belong Lortat-Jacob's four cases in which the operation (resection of a total longitudinal strip of muscle 2 cm. wide, extending from the aortic arc to the cardia) was carried out because of esophageal dyskinesia or diffuse spasms of the thoracic esophagus.

Such a classification errs perhaps in excessive formalism, it presents, nevertheless, a certain practical utility, since to each of the three groups there often corresponds a particular clinical and radiologic case, and, in consequence, particular therapeutic methods.

Microscopic Appearance—The characteristic structure of these tumors is represented by little bunches of smooth muscle fibers, which course along with out any order, they are located first haphazardly, then longitudinally, now transversely, now obliquely. Vortical placing, characteristic of tumors of an analogous structure in other organs (the uterus, for example), has not been noted in these leiomyomas.

The fibrocellular nuclei, narrow and long, are rounded at the ends. Oval, rounded, or spindle-like shapes, however, are not unknown.⁶

The protoplasm may appear smooth, homogeneous, or with extremely fine striations (Bezza was able in one case to distinguish the myofibrils).

The connective tissue is sometimes particularly ample. We speak in such cases of leiomyoma fibrosum or of fibroleiomyoma, often the little muscle bunches have very fine connective fibers permeating them, and connective elements are at times present between the individual muscle cells.^{3, 11} According to Bezza,⁶ a net, which is collagenous in nature, exists effectively around each single fiber, but a connective tissue would be present between cells only in particular conditions. Abundant elastic fibers are sometimes attached to some myomatous tumors, especially if pedunculated. The vascular element is generally scarce, vascular lacunae are often recognizable rather than true and proper vessels. Bezza, in his five cases, found fissures with an endothelial wall, lacking red

corpuseles, very probably interpretable as lymphatic lacunae. We can often observe degenerative and necrotic phenomena, and calcareous deposits visible sometimes even to the naked eye.

In "widespread leiomyomatosis," the nodules (more or less distinguishable) are contiguous and appear in rosary form. Composed of muscle cells of variable thickness and size which intersect each other with order, they frequently reveal an interstitial infiltration of an inflammatory nature.

'58

22 58



Fig 1



Fig 2

Fig 1—Case 1. Preoperative esophagram demonstrating displacement to the right in lower third of esophagus and concave appearance of its left wall. The arrows indicate the tenuous rounded shadow of the tumor mass.

Fig 2—Another esophagram of the same case.

CASE REPORT

CASE 1—M. P. Aged 54 years, was hospitalized June 11, 1949.

Operative diagnosis: Tumor of the esophagus (lower third).

Case history: One year prior to admission the patient suffered morning attacks of vomiting. Three years prior to admission he had had a similar vomiting episode of ten days' duration. Roentgenogram examination revealed minimal esophageal stenosis. From July to October 1948 he underwent medical and roentgen therapy in the Montreal Hospital. No weight loss was ever noted.

System review: Physical and laboratory examinations were normal.

Respiratory system: There was nothing abnormal in the chest. Clear plexus sound was heard.

Endoscopic examination of the alimentary canal: Esophagus was slightly enlarged. In its lower part no evident mucous changes were noted in addition to irregularity and rigidity in its left outline which showed a slightly concave appearance as from extrinsic pressure. It was displaced to the right and back. In this location there was a tenuous

shadow, well defined laterally (medially it was confluent with the esophageal wall on which it would seem to have attached itself), and which could be ascribed to a tumorous process. The opaque passage through the terminal esophagus was not sensibly obstructed, even though a slight bulge was noted above.

Conclusions There were organic changes in character of the terminal esophagus, ascribable to a neoplastic process in the left lateral wall, development chiefly external, but also intramural. Esophageal lumen adequate.

Operation (By Professor Forni, March 13, 1949) Endotracheal general anesthesia was administered. Thoracophrenolaparotomy (subtotal resection of the seventh rib) was performed showing total pleural and partial sternopericardiac symphysis, which were freed

Fig 3



Fig 4

Fig 3—Case 1 External appearance of the tumor sinuous in form with characteristic lobulation
 Fig 4—Case 1 Section of the tumor

Fig. 5

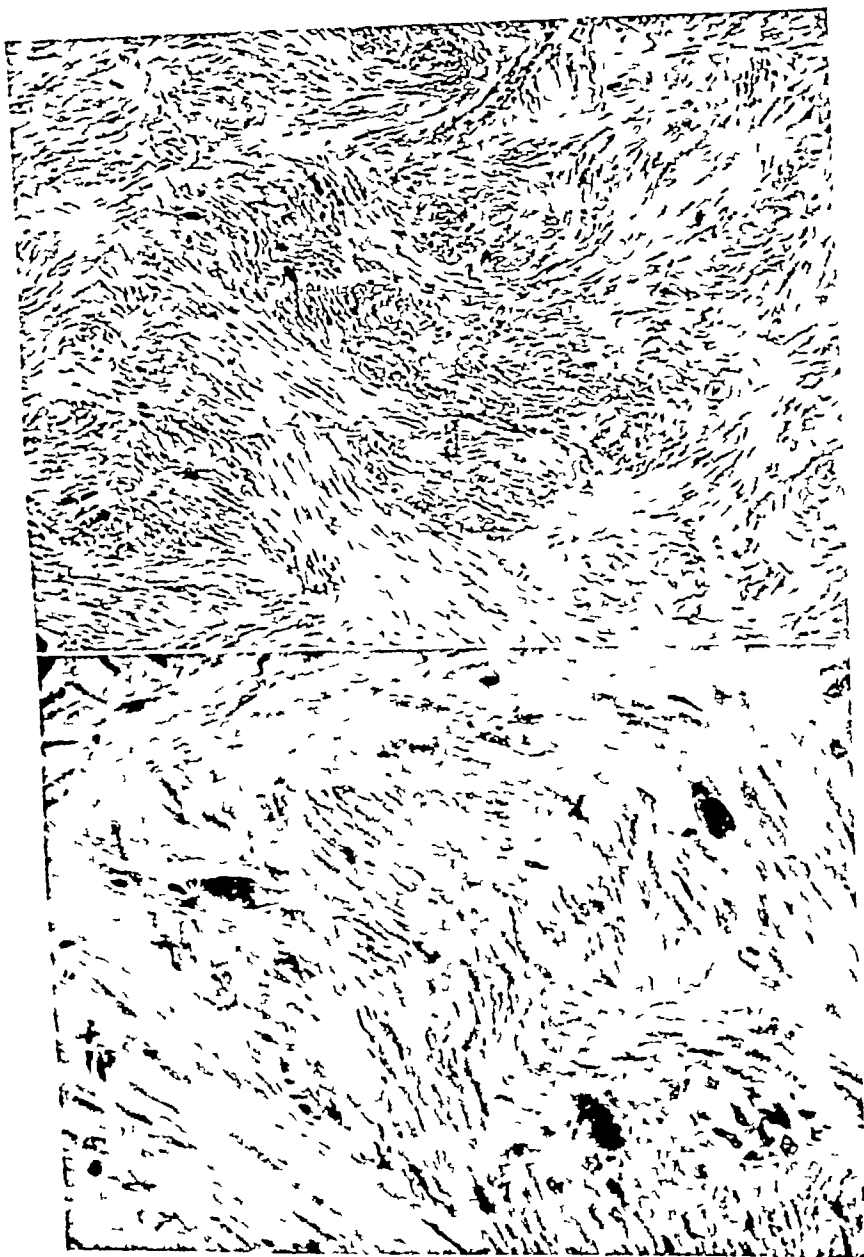


Fig. 6

Fig. 5.—Cut. 1. Photomicrograph of the tumor, demonstrating various groupings of muscle cells, affected by Inselsten (Van Gieson stain, $\times 67$).

Fig. 6.—Cut. 1. High power photomicrograph of the tumor ($\times 320$) of the outstanding (in cut. 1) the degenerative ones are particularly noticeable.

The diaphragm was incised and the esophagus mobilized, at which point a tumor, folded in U form, was found in the wall thickness, which bulged considerably from under the adventitia. The adventitia was incised, and the procedure for isolating the tumor, which was about 9 cm in length, initiated. During the enucleation process the mucosa was opened at two limited points. The tumor appeared to be of an irregular spindlelike form, with the U shaped folding described, it was elastic in consistency, yellowish white in color, lobulated, and probably of connective tissue structure. Triple layer closure of the esophagus was done, thorax was closed without drainage, penicillin was used endopleurally and intramurally.

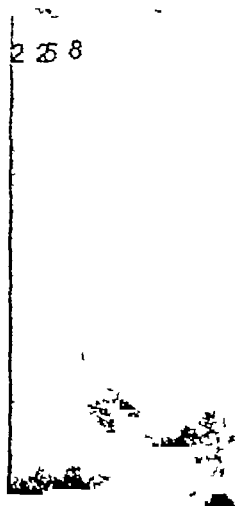


Fig 7



Fig 8

Fig 7—Case 1 Postoperative esophagram six months after demonstrating good esophageal canalization

Fig 8—Case 1 Postoperative esophagram twelve months after demonstrating the continuance of the esophagus in good working order

Histologic report The tumor was composed of tissue, compact in structure, and bunched in type. Such tissue appeared sometimes richer, sometimes poorer, in cellular elements, disposed in rectilinear or undulating bundles, variously placed, now diverging, now intersecting. The stroma and the scarce intercellular substance were composed of delicate collagenous homogeneous fibrils with here and there a slightly hyaline appearance. Vessels mostly very small in size, were running through this stroma and presented almost one endothelial wall only, they seemed to be intimately interwoven into the tissue, with some lymphomonocytic element in the adventitia.

The cellular elements which composed the tumorous tissue did not present a uniform structure. For the most part it proved to be of greatly elongated cells, whose cytoplasm slightly eosinophilous, had somewhat uncertain limits and was characterized by a certain longitudinal fibrillar disposition, disappearing at the poles. The nucleus was slender in form, at times a little curved as a comma threadlike or tortuous. Larger oval nuclei were sometimes seen with the chromatin in a hyperchromic homogeneous manner, the limits were not sharp and gave the impression of a swollen and worn tissue. For the most part such appear

inlets coincided with the regressive phenomena of the cytoplasm and with the formation of cytoplasmatic lumps. Other elements were more intimately interwoven in the collagenous tissue.

With the exception of the mentioned appearance (sometimes gigantic) of nuclear regression neither karyokinesis nor signs of abnormal proliferation were noted.

A trichrome stain (Masson, Van Gieson) showed the muscular nature of the tumor distinctly.

Histologic diagnosis Leiomyoma

Postoperative course The patient had an uneventful convalescence. After two weeks the wound was well healed and the patient was eating without difficulty. Barium swallow revealed a satisfactory anastomosis. The patient was discharged March 30, 1949, and continued his normal life in Canada completely free of any complaints. He died three years later from cardiac insufficiency and acute concomitant pulmonary affection.

This case offers some interesting clinical and histopathologic consideration. Of particular interest was the prolonged symptomatology, which was, in fact, very unusual and limited to vomiting attacks in the morning prior to eating without other characteristic signs of esophageal stenosis. The tumor mass as shown in the roentgenogram, was at first considered to be of a malignant nature so much so as to warrant a course of roentgen therapy. The prolonged symptoms, their lack of progression, the unchanged body weight, the good general condition, and an esophageal lumen clinically and radiologically maintained suggested, however, a benign process confirmed by the operation and histologic report.

The prominent regressive phenomena, which characterized the microscopic findings, and which have already been described by other authors, are worthy of note. Ribbert found on the cell periphery the presence of hyaline bodies which he considered to be parasites. Houette excluded such a hypothesis but maintained the little hyaline lumps were caused by precipitation of the protoplasm for changed colloidal equilibrium of the cellular protoplasm. More recently, Bizza declared such appearances to be regressive phenomena, in the sense of microfibril fragmentation and decomposition.

In our case and in contrast to the findings of other authors, the degenerative phenomena chose to affect the nucleus, probably as a result of the roentgen therapy carried out six months prior to the operation. Such a finding might acquire a notable significance, since descriptions of leiomyomas enucleated after roentgen therapy have not been made.

Case 2— E, aged 21 years, was hospitalized March 4, 1953.

Preoperative diagnosis Tumor of the esophagus (lower third).

Case history The tumor in question was discovered by chance in the course of a recent roentgen examination one month prior to hospitalization necessitated as a result of about a three month persistence of slight asthenia, lack of appetite, postprandial epigastric swelling, and pyrosis. The patient attributed these sensations, which had appeared in previous years in a mild form and for brief periods to his particular type of work (in a factory where the air was saturated with dust and chemical vapors) and to the pressure of the shoes on his epigastrium while working on the lathe.

The patient experienced no other disturbances—no dysphagia, no regurgitation, and no pain.

Physical examination System review and laboratory studies were negative.

Radiologic examination of the alimentary canal Esophagus was slightly dilated, especially in its lower part where two luminary forms were noted about the size of a hazelnut, well defined in the epigastric site. The mucous folds were soft and the opaque passage into the stomach was not obstructed.

The stomach of hook form, was normal in size, empty prior to eating, and without organic form. There was complete peristalsis on both curvatures. Duodenal bulb was regular. Second portion was normal in appearance. Time of emptying of the stomach was within physiological limit. Passage through small bowel was normal.

Postoperative course Intracystic benign neoplastic process affecting the epicardial section, polypoid in type.

Esophagoscopy Esophagus was normal in form, size, and consistence as far as mid third. Lower third appeared slightly dilated with the mucosa stretched out. In the area immediately above the cardia two roughly rounded formations, which were pendulous, completely covered by mucus and not bleeding, were observed. One of these was situated on the left on the anteriolateral aspect, the other slightly lower on the right, on the posterolateral. Firm in consistency, not very elastic, they did not obstruct the passage. Diaphragmatic movements lifted the two formations synchronously.

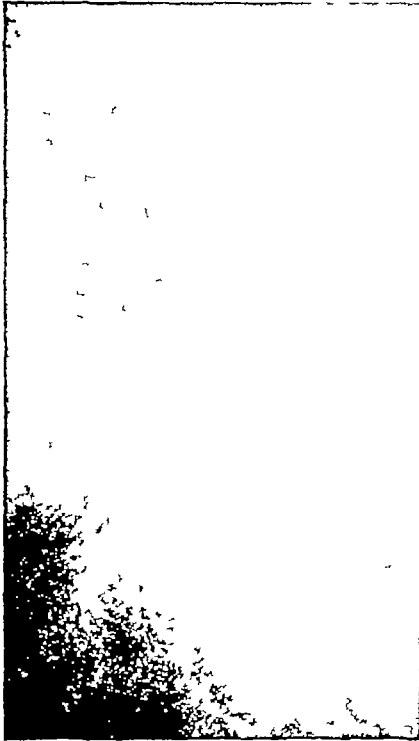


Fig 9

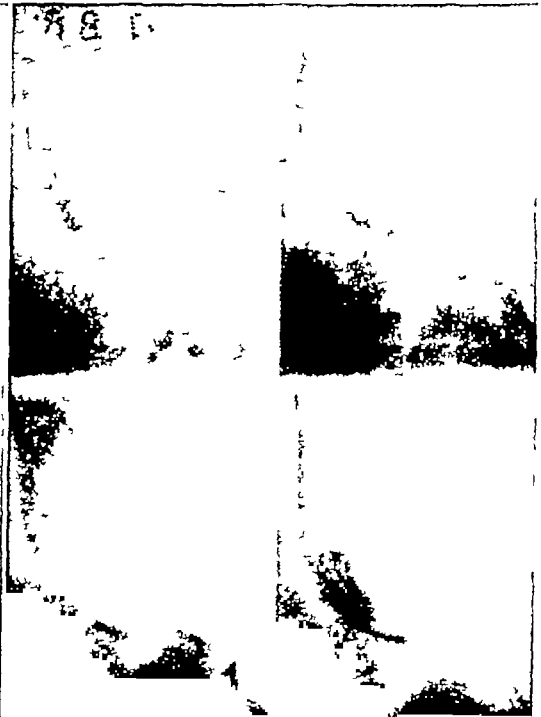


Fig 10

Fig 9—Case 2 Preoperative esophagram demonstrating dilation in lower third of esophagus the presence of two lacunary images and the regularity of the mucous folds

Fig 10—Case 2 Another esophagram of the same case

Operation (By Professor Forni, March 28, 1953) Thoracophrenolaparotomy was performed. By incising the mediastinal pleura and dissecting the triangular ligament, the esophagus was taken out through the hiatus. It was isolated for an area of about 10 cm. In the wall a slightly knobby neoformation presented itself, which extended about 7 cm in length. At the cardia it took on a U shape and then rose into the opposite wall in a space of about 4 cm, taking on a squat, but ever knobby appearance. The muscle wall was incised, and the shelling out of the tumor, which was surrounded by a small layer of adventitia, was begun. The process of enucleation was carried out without entering the mucosa except in an area of some millimeters, where the apex of the tumor joined with the mucosa, which was closed with a suture. The finding was analogous to that in the preceding case in regard to shape, size, and appearance.

Histologic report The fragments, taken at various points from the little mass, presented a bunched structure throughout. The tumor consisted of currents of cellular elements, variously set, greatly elongated, some of which were disposed in a parallel and, at



FIG. 11—Case 2. External appearance of the tumor showing lobulation and U shape.



FIG. 12—Case 2. Section of the tumor

Fig 13

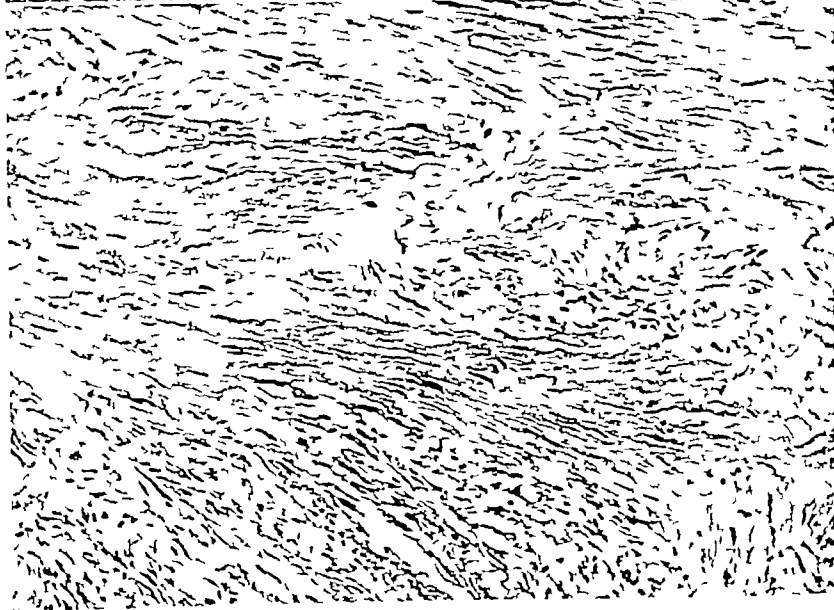
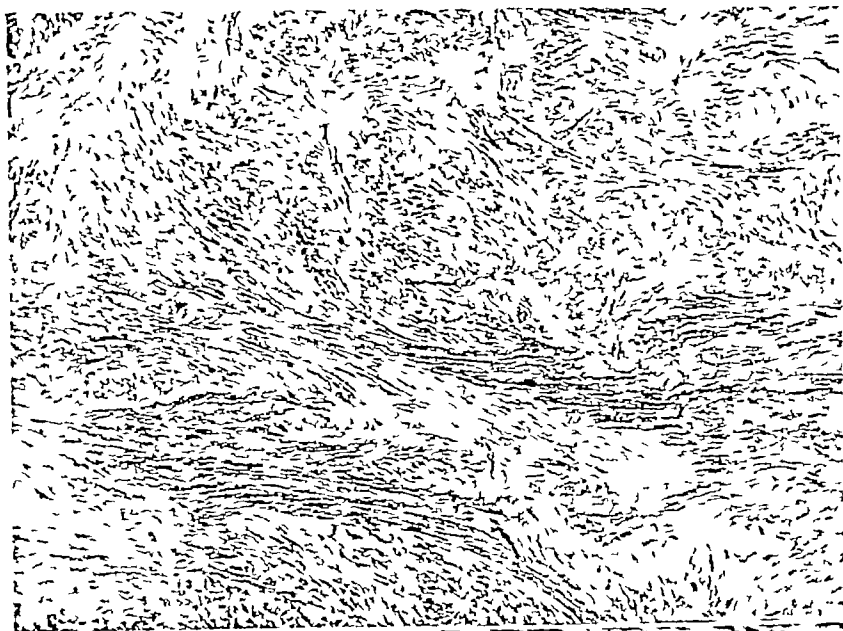


Fig 14

Fig 13—Case 2 Bunched structure of the tumor determined by currents of fibrous muscle cells variously situated ($\times 80$)

Fig 14—Case 2—Enlarged detail of Fig 13 showing the longitudinal striation and the spindle- or threadlike appearance of the nuclei ($\times 319$)

the same time, sinuous manner. Sometimes too, these cells were grouped in large vortices, so that the microscopie section caught the cell bundles in various incidental groupings.

Some fine fibrillar septa united the bundles, which were particularly dense around the vessels, losing themselves in the muscle wall of the latter.

The cytoplasm also was sometimes not clearly distinct from the fibrillar stroma. Where it was better preserved, it was possible to distinguish a longitudinally striated structure and a considerable eosinophilia, with some swelling here and there. The differentiation between the cellular elements and the stroma which appeared otherwise run together, made itself particularly evident with the elective staining (Mallory, Van Gieson).

The nuclei were spindlelike, sometimes curved in common form, sometimes somewhat undulating or stretched out in threadlike appearance. The chromatin content of such nuclei was regular and finely powdered, with some little nucleoli here and there.

Histologic diagnosis Leiomyoma

Postoperative course The postoperative course was uneventful. The patient was eating a full diet on the tenth postoperative day and had a well healed wound.

In this patient too, the symptoms, although minimal, had been in existence for some years, characterized by long periods of remission and by a complete absence of dysphagia, regurgitation, or pain. The only sensations were moderate asthenia, lack of appetite, and at times postprandial epigastric swelling and pyrosis.

Esophagoscopy and roentgenograms demonstrated the existence of the tumor, the prevalent development within the lumen and polypoid characteristics, the mucous integrity, the probable benign nature of the tumor, and its probable origin in the muscle wall. The operation confirmed such impressions, producing a tumor with characteristics analogous to those in Case 1 but with a tendency to development toward the esophageal lumen, as to distend and notably thin the mucosa, and to take on at some points a polypoid appearance.

The histologic examination demonstrated the same bunched structure, as in the preceding tumor, and the same cytoplasmic and stromal characteristics, but showed complete absence of any aspect of nuclear regression.

CLINICAL COMMENTS

Esophageal myomas are frequently asymptomatic. Of 95 cases in the literature 61 were free of clinical symptoms.

The appearance of symptoms is sometimes preceded by badly defined disturbances or painful sensations which the patient is often unable to describe, or which are sometimes considered to be of a functional nature, as by Lanzara.⁴⁴

Dysphagia is a very frequent isolated symptom,^{3 15 44 49 61 71 82} but more often accompanied by other disturbances. It is generally found with ingestion of solid foods and rarely with liquids. In the case referred to by Lanzara⁴⁴ the patient to feed himself, was obliged to recur to Cohn's maneuver (rapid swallowing of a glass of water after every mouthful of food). Sometimes dysphagia accompanies only certain foods, as in Cornell and Shehady's patient¹⁹ (dysphagia with sweet, cold, or acid foods). Very often it appears at irregular intervals with more or less rapid regression. It rarely shows a course constant or progressive to the point of stenosis.^{15 4 66} It can persist for years, as follows: twenty-four years,¹ seven years,⁵⁶ and six years.⁴⁴

Schäfer and Kittle⁴ hold that benign tumors causing dysphagia are rarely extramucosal in origin (myomas), but that their origin is more often submucosal or mucosal since these last pedunculate easily, intruding into the esophageal lumen. Dysphagia secondary to leiomyoma, according to the same authors, should be due either to great enlargement or to malignant transformation. From the literature however it is inferred that dysphagia is present also in small sized tumors.^{2 44 61 6}

Pain during deglutition, particularly of solid food, has been described only by Garlock³⁰ Frequently the pain is, instead, spontaneous, or begins immediately,⁷¹ or about ten minutes after eating³⁷ It is more frequently retrosternal,^{30 34 39 71} but can also be found in the thorax,^{52 74} in the epigastrium,^{23, 2} and even in the shoulders³⁰ It can be continuous, but more frequently is crampy with spontaneous remissions and exacerbations

Sometimes nausea and vomiting^{23 45} are present, or direct alimentary vomiting takes place^{9 30 37, 71, 74} Other effects are regurgitation,^{30, 44, 45} a sensation of weight in the epigastric region,^{9 19 37} and dyspeptic sensations, in which periodic remission of the symptoms makes one suspect the presence of an ulcer⁴ No author has found sialorrhea

Sometimes there can be loss of weight, depending on the grade and duration of the dysphagia If this regresses, the recovery of body weight can be evident in a short time⁴⁵ The general state of health is, however, generally well maintained, even many years after the beginning of the illness

There have also been descriptions of symptoms of compression of near by organs (trachea and bronchi) with dyspneic crises⁶² which, nevertheless, might be independent of stenosis Dyspnea associated with coughing is present in Manzocchi's case⁵² (Leiomyoma of the lower third of the esophagus came to resemble an irritation of the vagus nerve)

The fundamental characteristic of the symptoms explained is their notable duration—sometimes for years—and their capricious evolution

In two patient⁴⁵ hemorrhage, which showed itself with hematemesis and melena, was experienced Lippich described perforation of the trachea by a tumor, whose muscular nature was not, however, histologically documented

DIAGNOSIS

In leiomyomas of the esophagus a positive clinical diagnosis is not possible In fact, dysphagia, retrosternal or epigastric pain, the sensation of weight, nausea, and vomiting are not characteristic, but common to all tumors of the esophagus and periesophageal spaces

A diagnosis of probability is possible,^{10 23 37 38 45 71} and, in our second case in addition to an accurate differential diagnosis by x-ray examination and esophagoscopy, completed, if necessary, by biopsy This last is, however, not always advisable, because of the necessity of penetrating the integral mucosa to reach the tumor and because of the possibility of creating wounds and trauma, which might prove serious

The radiographic image of these neoplasms presents appearances different in accordance with their anatomic form

In the rare cases of pedunculated leiomyoma, one can perceive a particular shadow which cuts into the esophageal lumen and which coexists generally with a wall which is functionally intact

In the intramural forms or in those where the tumor arises in the periesophageal space, a simple roentgenogram can demonstrate the presence of a rounded shadow, more or less opaque, with clean edges, and well defined in respect to the surrounding tissue In the barium swallow, the tumor mass is confused with the esophageal wall There often exists, at the same time, dilation

and deviation of the esophagus, whose lumen generally presents deformations (Case 1) The opaque meal passage is not sensibly obstructed, although a slight stoppage above is to be noted

Harper and Tisceno³⁷ tried to enumerate the most important radiologic characteristics, which are useful mainly in the differential diagnosis with carcinoma of the same site The authors underlined the importance of a tumor mass, for the most part not fixed, connected with the deformed esophageal area and more or less swollen in the mediastinum They recognized the frequent enlargement at the same level of the tumor, or a protrusion of that part of the wall on a level with and opposite to, the tumor, the absence of undermined margins, the variations in the form and size of the refilling defect, the absence of any ulceration of the mucosa corresponding to the neoplasm, and the conservation of the normal fold in the immediate neighborhood of the refilling defect

Recently Chalnot and co workers¹⁰ underlined the importance, after a barium swallow, of some aspects of the lacunar image, which, characterized by limits more or less regular, frequently reproduces lobulation typical of leiomyomas covered by integral mucosa and presents at its extremities two little spurs in relation with the superior and inferior poles Such signs indicate the intactness and softness of the mucosa and are opposed "to the rigid, fragmentary, and irregular appearance of the mucosa affected by a malignant neoplasm"

Lortat-Jacob¹⁰ noted a kind of arborescent image, in a leiomyoma with a macroscopic appearance similar to our first case, without amputations, due, in his opinion, to the barium filling up the crevices provoked by the lobulations of the tumor The same author has also reported a flattening of the esophageal profile, which reminds one of ureterographical images for a ureter flattened like a sword blade Such an appearance would be due to the particular and frequently noted horseshoe form of the tumor

Of particular moment in the two personal cases are the enlargement and the displacement of the area of the esophagus affected by the tumor, the presence of convexity as a result of extrinsic pressure, the existence of a regular-rounded shadow the uniformity of the mucous layout, and the complete absence of obstruction in the barium passage

A careful analysis of the radiologic findings (before or after barium meal), especially if combined as a result of repeated tests, can permit within a limited field of error not only the generic diagnosis of a benign tumor of the esophagus, but the ascertainment of the eventual preponderant development (intrinsic or extrinsic) assumed by the tumor mass

Esophagoscopy produces good results in pedunculated tumors, whose size, shape, location, and origin can be evaluated with certainty

In intramural leiomyomas or in those which develop in the periesophageal area an intact mucosa is noted more or less raised by the underlying tumor Sometimes the mucosa appears congested¹ or superficially eroded³⁰ The esophageal lumen at the level of the tumor can be restricted so as not to permit the passage of the esophagoscope¹ in the overlying esophageal area dilatation usually in a limited degree, is frequent "4"

During esophagoscopy, it is sometimes possible to take up a fragment of tissue for a histologic examination which alone can permit a definite diagnosis. We have previously hinted at the obstacles to this procedure, and at the necessity of preserving intact the mucosa, which is often extended and very friable.

DIFFERENTIAL DIAGNOSIS

Of the tumors which recur frequently in the differential diagnosis with leiomyoma of the esophagus, let us consider above all carcinoma, sarcoma, the other benign neoplasms, megaesophagus, and cardiospasm—in short, the neoplasms and malfunctions located in the periesophageal area and compressing the esophagus.

Carcinoma in general presents a symptomatology briefer in duration and an evolution which is progressively fatal. Esophagoscopy shows, in the ulcerated forms, the loss of substance and edges slightly raised, and in the vegetant ones a polypoid or fungus appearance, a notably hard bulge in the infiltrating types, which tends to compress the lumen. Bleeding, moreover, is easy.

Sarcoma is more often characterized by a rapid evolution, the absence of linear infiltration frequent in the epithelium, and the not infrequent pulmonary, pleuromediastinal, and tracheobronchial metastases.

The differential diagnosis, based above all on the sometimes prevalent copious esophageal dilation above the leiomyoma, is also valid for esophagospasm and megaesophagus.^{44 45}

For the diagnosis of cardiospasm x-ray findings are necessary of the image of the esophagus in cigar form or of the cardiac channel as a root or rat's tail, and esophagoscopy images with an obstruction in the passage of the instrument which proceeds slowly but completely.

Abundant regurgitation, a postprandial sensation of obstruction which passes sometimes suddenly with a special noise, dysphagia, and retrosternal oppression which tend to increase progressively, bad oral taste, and the characteristic sensation of entry in the cavity of the esophagoscope, once cricoid restriction has been overcome, incline one toward the diagnosis of megaesophagus. The diagnosis is confirmed by the radiologic examination, which shows an esophagus notably dilated in sack form, with pockets, or cylindrical in shape, in addition to being considerably elongated and sinuous, and the constant integrity of its subdiaphragmatic portion.

In so far as concerns the extraesophageal compressions, the tumors which can recur in the differential diagnosis are listed in a simple manner for purposes of brevity: (1) aneurysm of the aortic arch in its posterior position and of the descending aorta, (2) aneurysm of the left subclavicular artery, (3) bronchogenic carcinoma, especially of the left main bronchus, (4) cystic tumor, or parasitical (*echinococcus*) or congenital (dermoid) cysts of the mediastinum, (5) thyroid or retrosternal parathyroid tumors, (6) lymphoglandular tumefactions (lymphosarcoma, lymphogranuloma), (7) folded esophageal diverticulum, modified in its radiologic appearance by state of repletion (saliva, food), (8) diaphragmatic hernia.

THERAPY

The therapy of leiomyoma of the esophagus is exclusively surgical. Monod¹⁰ and Dubois have carried out a ligation of the pedicle of an esophageal tumor arising into the mouth. In the rare eventuality of a pedunculated leiomyoma, its removal can be carried out orally during esophagoscopy. Almost always, however, the leiomyoma (intramural or subadventitial) requires thoracotomy, generally from the left, but also from the right (Rossello's and Chalmot's cases were localized in the higher mid-third). The method known as thoracolaparotomy,^{11 12 16 23 37, 41 61 74 83 86 87, 89 90 91 93} is used if the tumor is located in the lower third or if it affects the cardia.

The esophagus having been exposed the tumor is seen in its characteristic appearance. Attachments with the nearby organs and the mediastinal pleura, infiltration, sclerosis or wall retraction as frequently seen in malignant tumors, do not generally exist.

The neoplastic mass, sometimes lobulated with serpentine prolongations which are more often rounded, appears in strict connection with the muscle membrane of the esophageal wall, and is, for the most part, well enucleable.

The method, followed by a number of workers,^{13 16 33 37 47 61, 74} consists in a prudent and progressive enucleation of the tumor without entering the mucosa. The mucosa can accidentally be opened in some limited points, however this has no great importance.

Enucleation is the operation of choice for single-node leiomyomas, but can also be performed for those with multiple nodes when good possibilities of leaving the mucosa intact exist. It presents no particular surgical difficulties or risks, and permits the esophagus to maintain its normal physiologic appearance.¹⁵

Those leiomyomas which cannot easily be enucleated by reason of their strict continuity with the muscle of the esophageal wall or because they are intimately attached to the mucosa and those of the cardioesophageal region (which frequently also affect the gastric wall) may require esophageal or gastroesophageal resection, according to the case in question.^{9 32 38 42 43, 52 66 71 76 78} This is followed, respectively, by end-to-end esophageal esophagostomy or gastroesophageal anastomosis.

Only three patients have died in the postoperative period after such a course of action. The long term results in general, have been satisfactory in the repeated control tests, with a good recovery of esophageal canalization. Some rare and temporary spasms may, however persist, or peptic esophagitis may show itself as in Fortat Jacob's patient.⁴⁰ Resection is accompanied by greater risks and sometimes by complications in gastroesophageal canalization, considerations which merit accurate evaluation when one is dealing with benign tumors which should require only in cases of necessity operations other than simple enucleation.

CONCLUSIONS

Leiomyoma of the esophagus is a tumor which is well defined from the anatomopathological and clinical points of view, notwithstanding its rather rare appearance (102 cases from 1797 to 1954).

It is most frequently found in the lower third of the esophagus. It more often affects the male sex (66.6 per cent) after the age of 50 years. Esophageal leiomyomas in the young are not rare.

According to the best opinions, the tumor originates from the muscle layers of the esophageal wall.

The most frequent anatomic form to appear in autopsy cases has been that of a single node, while in clinical cases the multinodular form has predominated.

Symptoms are common to the tumors which narrow the esophageal lumen. Generally they are of long duration, but without affecting the general well being—a characteristic moreover, of all benign neoplasms.

Since diagnosis is not always possible merely by evaluation of the symptoms, the importance of such diagnostic aids as radiology, esophagoscopy, and sometimes biopsy, must be emphasized.

The clinical course is, for the most part, slow. Evident findings of malignant transformations have only once been mentioned.

The only efficacious treatment at our disposal is surgical (most frequently enucleation of the tumor without opening the esophageal mucosa), a therapy that leads to really satisfactory immediate and long-term results.

SUMMARY

Two case reports of leiomyoma in the lower third of the esophagus are presented in men of 54 and 21 years, respectively, operated on in the Surgical Clinic of Bologna.

The enucleation of the tumors was followed by recovery, controlled by repeated radiologic examinations (good esophageal functioning).

Such experiences and a survey of the literature on the subject lead to a description of the principal anatomopathological, and clinical characteristics of leiomyoma of the esophagus with therapeutic and prognostic deductions.

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INTRABRONCHIAL LIPOMA

A CASE REPORT

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THE increasing availability of thoracic surgery, bronchoscopy, and then diagnostic adjuncts is reflected in the rising number of reported benign intrabronchial tumors. The lipoma ranks third in incidence among benign mesodermal intrabronchial tumors, but comprises a very small percentage of them.¹ Its rarity may be judged by the fact that we have been able to find only fifteen previously described surgically removed and histologically confirmed intrabronchial lipomas or fibrolipomas. Of these nine were reported prior to 1950 whereas six have been reported since that time. The illustration of intrabronchial lipoma in Liebow's fascicle¹⁵ is from an unreported case operated on in 1945, to the best of our knowledge.¹⁶

Rokitansky is credited in previous reports^{5, 14} with describing the first intrabronchial lipoma in 1854, discovered at necropsy. The first reported surgically removed lipoma was that of Kernan in 1927.¹ During the next twelve years three closely related tumors, fibrolipomas, were reported by Myerson in 1929,² Wessler and Rabin in 1932,³ and McGlade in 1939.⁶ These tumors, along with others reported are listed in Table I.

TABLE I. REPORTED CASES OF INTRABRONCHIAL LIPOMA, SURGICALLY TREATED

AUTHORS	YEAR	SEX	AGE	COLOR	LOCATION	TREATMENT
Kernan ¹	1927	M	50	W	Right main bronchus	Bronchoscopic excision
Myerson ²	1929	M	47	W	Left main bronchus	Bronchoscopic excision
Jackson & Tuckson ⁴	1932	F	64	W	Left main bronchus	Bronchoscopic excision
Wessler and Rabin ³	1932	M	48		Left lower lobe	Bronchoscopic excision
McGlade ⁶	1939	M	48	W	Left main bronchus	Bronchoscopic excision
Vinson and Penbleton ⁵	1942	F	29	N	Left main bronchus	Bronchoscopic coagulation (pneumonecotomy advised)
Watts, Claggett and McDonald ⁸	1946	M	51	W	Left upper lobe bronchus	Left upper lobectomy
Whalen ⁹	1947	M	61	W	Left upper lobe bronchus	Bronchoscopic excision
Fell ¹⁰	1949	M	40	W	Right main bronchus	Bronchoscopic excision
Carlini, Lewis and McDonald ¹¹	1951	M	52	W	Left upper lobe bronchus	Left upper lobectomy
Sou and Foucault ¹²	1953	M	75	W	Right lower lobe bronchus	Bronchoscopic excision
		M	79	W	Right upper lobe bronchus	Bronchoscopic excision
Beuten and Hattvis ¹³	1952	M	76	N	Left main bronchus	Bronchoscopic excision
Semart ¹⁴	1953	M	65	W	Trachea	Bronchoscopic excision
		M	69	W	Left main bronchus	Bronchoscopic excision

From the Jackson Valley Community Hospital, Kingsport, Tenn.
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The occurrence of these lipomas in three Negroes in a total of sixteen may be a chance incidence of no statistical significance, but, if such a high proportion continues as the number of reported cases increases, it will be an interesting contrast to the lower incidence of bronchogenic carcinoma in Negroes than white men. The general preponderance of men over women with pulmonary tumors exists in this series as well. More of these lipomas have been reported in the left bronchus than the right. It may be that this is due to the fact that they are all in the major bronchi, either stem bronchus or primary divisions where fat is normally found, and the left main bronchus exceeds the right in length, giving a greater chance of occurrence here. The lower reported incidence of lipomas in the trachea is difficult to reconcile in view of the greater proportional length of this airway, and indicates the operation of factors other than chance in the development and location of these tumors.

The growth characteristics reported indicate that the intrabronchial lipoma is often pedunculated, occasionally sessile, and in one instance in dumb bell distribution that had a deep portion external to the cartilaginous plates.¹¹ Thus, in the last instance, the intrabronchial lipoma resembles the pattern seen in bronchial adenomas, and confirms by its continuity the hypothesis that these tumors arise from normal fat located external to the cartilage. The polypoid nature of the tumor is reflected in the frequency of bronchoscopic removal. Pulmonary resection was thought to be indicated because of distal suppurative disease in only three of the previously reported cases. It was actually performed in two. The lipoma, like other histologically benign tumors, may produce serious and prolonged illness, even death, when located in such a critically important position as a main bronchus.

The following case report demonstrates a common sequence of events seen in benign intrabronchial tumors, namely, partial then complete respiratory obstruction with distal suppuration and bronchiectasis. Hemoptysis, which occurred in this case, was mentioned in five of the previously reported cases.

CASE REPORT

This 60 year old Negro man was first seen in the outpatient department on April 10, 1951, with the presenting signs and symptoms of pain in the left chest, cough, cyanosis and dependent edema. During the preceding two or three months he had noted shortness of breath on exertion, orthopnea and had had several bouts of hemoptysis. His appetite remained good, and there had been no known weight loss. He was hospitalized one week later for study of possible carcinoma of the left lung.

The principal findings on examination at this time were: temperature 98.6° F, pulse 100, blood pressure 140/80, respirations 20. He appeared to be about the stated age, and moderately well nourished. There was dullness to percussion over the lower left chest with breath sounds diminished to absent over this area. Crepitant râles were present over the remainder of the left lung and the base of the right lung. The heart was enlarged to the left, as shown by percussed dullness and x-ray examination (Figs. 1 and 2). No peripheral edema or cyanosis was noted. The liver and spleen were not palpable. A blood count showed 4.5 million red blood cells, 13.5 Gm hemoglobin, 12,800 white blood cells with 62 per cent neutrophils, 35 per cent lymphocytes, and 3 per cent eosinophils. Urinalysis was normal. Serologic test for syphilis was negative. Nonprotein nitrogen was 55 mg per cent. Sputum smears and cultures were negative for acid fast bacilli. An electrocardiogram showed left heart strain and digitalis effect.



Fig 1

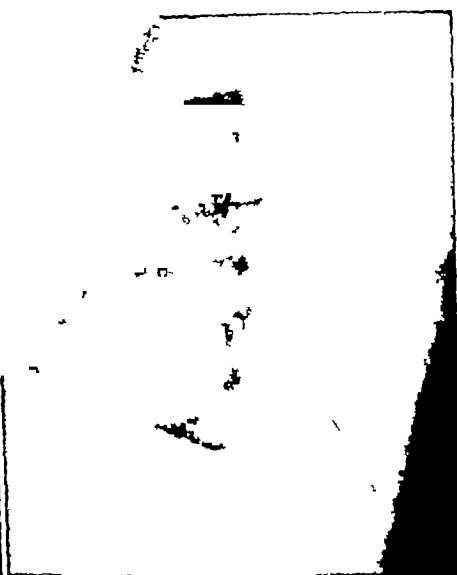


Fig 2

Figs 1 and 2—Anteroposterior and lateral films of chest on first admission April 26 1951



Fig 3—Film of chest on Sept. 2 1952

The patient was discharged from the hospital on May 1, 1951, with the diagnosis of cardiac failure and consolidation of the left lower lobe of the lung. Treatment with antibiotics, diuretics, digitalis, aminophylline, and laxatives had produced improvement. The possibility of a pulmonary neoplasm was not considered further at that time. A repeat x-ray of the chest, done ten days after discharge, showed infiltration of the left base and emphysema of the left upper lobe.

He was next seen in the outpatient department on Sept. 2, 1952, with the same symptoms, but some loss of weight and strength. The entire left side of the chest was dull to percussion with diminished to absent breath sounds. He was readmitted to the hospital on Sept. 9, 1952, when he was first seen by one of us (R. E. M.). The cardiac condition appeared clinically improved in that the chest heart ratio was normal, there was no evidence of congestive failure, and the nonprotein nitrogen was 30 mg. per cent, however, the electrocardiogram still showed left heart strain and occasional ventricular premature beats. The increased consolidation of the entire left lung (Fig. 3) with mediastinal shift to the left but an improved cardiac status was noted. Other findings were essentially unchanged.

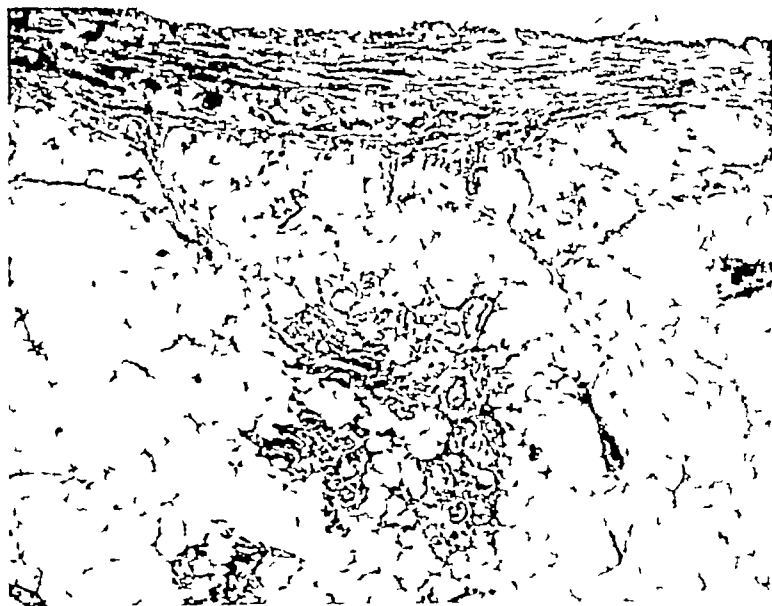


Fig. 4—Microscopic appearance of bronchial lipoma showing respiratory epithelium and inclusion of mucous glands.

Bronchoscopic examination was carried out the next day, at which time a smooth globular tumor was found to block the left main bronchus almost completely just distal to the carina. A biopsy of the growth showed normal respiratory mucous membrane with some underlying fatty tissue. Following this, the productive cough stopped and remittent fever ranging from 102° to 103.6° F. developed. This was thought to be due to complete obstruction from edema associated with the biopsy. An hematocrit of 33 was raised by transfusions to 41 in preparation for operation.

A transpleural bronchotomy was done on Oct. 2, 1952. The tumor, which had a narrow pedicle, was easily removed. A pneumonectomy with transection of the bronchus distal to the bronchotomy site was performed because of the extensive suppurative disease of the lung.

The tumor measured 2.5 cm in greatest dimension. It had a smooth multilobular surface, pale yellow color, and soft rubbery consistency. On cut section it was solid with a pale lemon yellow, slightly translucent appearance. Small lobular areas were partially surrounded by thin white septa. Microscopic examination showed it to be composed of mature adipose tissue with the inclusion of many mucous glands (Figs 4 and 5). The epithelium was denuded in some areas but, where present, was of respiratory type with areas of squamous metaplasia.

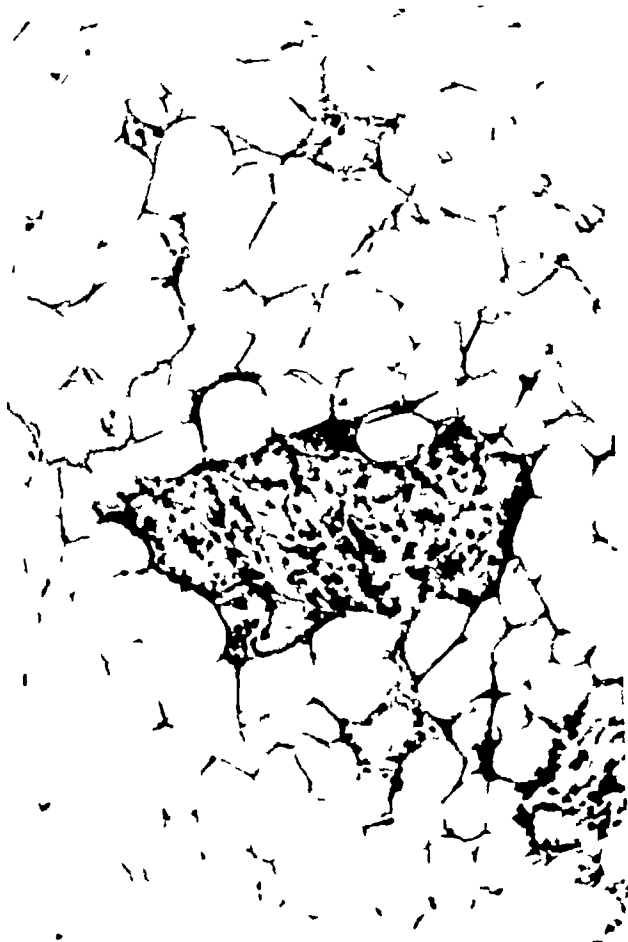


FIG. 5.—Microscopic appearance of lipoma showing mucous glands surrounded by adipose tissue.

The left lung, submitted separately, weighed 800 grams and was the seat of considerable bronchiectasis and fibrosis involving both lobes, with extensive acute and organizing focal pneumonia.

During the early follow-up period the patient remained well free of cough, and gained in strength and weight. He occasionally lapsed into congestive failure because he did not follow prescribed treatment but always responded well to hospitalization. He was most recently seen in congestive failure in March 1954. On bed rest, diuretics, and redigitalization he lost 10 pounds of edema. The x-ray appearance of the left thorax has been stable with no change in the level of the diaphragm or position of the heart and trachea. He left the hospital compensated at rest.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-FIFTH ANNUAL MEETING

APRIL 24, 25, AND 26, 1955

CHALFONTE-HADDON HALL, ATLANTIC CITY, N. J.

PROGRAM

Sunday Morning, April 24, 1955

8:30 A.M. Business Meeting

9:00 A.M. Scientific Session REGULAR PROGRAM

- 1 Pleurobiliary and Bronchobiliary Fistulae Herbert D. Adams, Boston, Mass
- 2 Staphylococcal Pneumonia in Infants A Report of Ten Cases W. E. Bloomer (by invitation), R. E. Cooke (by invitation), G. E. Lindskog, and S. Giammona (by invitation), New Haven, Conn
- 3 Management of Massive Hemoptysis, Not Due to Pulmonary Tuberculosis or Neoplasm. I. J. Ehrenhaft and Rodman E. Taber (by invitation), Iowa City, Iowa
- 4 The Significance of the Anterior Segment in Bronchiectasis. Richard H. Overholt and Wilford B. Neptune (by invitation), Boston, Mass
- 5 Spontaneous Rupture of the Esophagus. George W. B. Starke (by invitation), Boston, Mass
- 6 One Stage Pharyngo-esophageal Diverticulectomy I. D. Mortensen (by invitation), O. Theron Clappett, Herbert W. Schmidt and Howard K. Gray, Rochester, Minn

Sunday Afternoon, April 24, 1955

2:00 P.M. Scientific Session REGULAR PROGRAM

- 7 The Diagnosis of Acquired Valvular Disease by Left Heart Pressure Recordings. Don I. Fisher (by invitation), Pittsburgh, Pa
- 8 A Laboratory and Clinical Evaluation of Operations for Coronary Artery Disease David S. Loughmiller (by invitation), Cleveland, Ohio
- 9 Technical Factors Which Favor Mammary Coronary Anastomosis With Reports of Forty Cases of Human Coronary Artery Disease Thus Treated. Arthur Vineberg and William Buller (by invitation), Montreal, Que
- 10 Analysis of 50 Patients Treated by Mitral Commissurotomy Five or More Years Ago. Robert P. Glover, Thomas L. F. O'Neill and O. Henry Janton (by invitation), Philadelphia, Pa
- 11 Evaluation of Techniques for the Repair of Auricular and Ventricular Septal Defects. Earl R. Kay, Frederick S. Gross (by invitation) and Henry A. Zimmerman (by invitation), Cleveland, Ohio
- 12 Experiences With Surgical Repair of Atrial Septal Defects. Elton Watkins, Jr., and H. L. F. Gray, Boston, Mass

Monday Morning, April 25, 1955

8 30 A M Scientific Session THORACIC SURGICAL FORUM

- 13 **Anastomosis of Right Auricle to Pulmonary Artery in the Treatment of Tricuspid Atresia** Elliott S Hurwitt, Dennison Young (by invitation), and Doris J W Escher (by invitation), New York, N Y
14. **Autogenous Thoracic Aorta Grafts of Pericardium and Nylon Net Using a Thrombin Fibrinogen Coagulum** Richard H. Adler (by invitation), Denver, Colo
- 15 **The Lymphatic Drainage of Silver Coated Radioactive Gold Colloids Following Intra-thoracic Administration to Pneumonectomized Dogs** P F Hahn (by invitation), Robert A Matuska (by invitation), Robert I Carlson, Stewart H Auerbach (by invitation), and George R Meneely (by invitation), Nashville, Tenn
- 16 **Circumferential Suture of the Mitral Ring—A Possible Method for the Surgical Correction of Mitral Insufficiency** Julio C Davila, Robert G Trout, Bart S Iain, and Frank Mansure (all by invitation) Sponsored by Robert P Glover, Philadelphia, Pa
- 17 **Studies Upon the Physiological Function of the Human Vagus Nerve in Various Pathological Pulmonary States** Osler A Abbott, W E VanFleet (by invitation), E R Duschesne (by invitation), and A E Roberto (by invitation), Emory University, Ga
- 18 **Temporary Unilateral Occlusion of the Pulmonary Artery in the Preoperative Evaluation of Thoracic Patients. A Preliminary Report** Herbert Sloan and Joe D Morris (by invitation), Ann Arbor, Mich
- 19 **Coronary Perfusion for Longer Periods of Cardiac Occlusion Under Hypothermia** Marvin L Ghedman (by invitation) and Norman E Shumway (by invitation) Sponsored by F John Lewis, Minneapolis, Minn
- 20 **Blood Volume Studies in Thoracic Surgical Procedures Using Radioactive Iodinated Human Serum Albumin (BHAS)** Clifford F Storey, Charles G Foster (by invitation), and Thomas G Mitchell (by invitation), St Albans, N Y
- 21 **Induced Cardiac Arrest for Intracardiac Surgical Procedures** Conrad R Lam, Thomas Geoghegan (by invitation), and Alfredo Lepore (by invitation), Detroit, Mich
- 22 **Open Left Heart Surgery in Dogs During Cardiac Arrest Below 10° C Temperature With and Without Extracorporeal Circulation** Frank Gollan, Reginald Phillips, Jr, James T Grace, and Raymond M Jones (all by invitation), Nashville, Tenn
- 23 **Physiological Observations in Experimental Pulmonary Insufficiency** Robert G Ellison, Walter J Brown, Jr (by invitation), Elmer E Hague, Jr (by invitation), and William F Hamilton (by invitation), Augusta, Ga
- 24 **Experimental Transposition of the Great Vessels.** Sanford E Leeds, Morris M Culliner (by invitation), and Sherman H Strauss (by invitation), San Francisco, Calif
- 25 **Controlled Total Body Arterial Perfusion for Open Intracardiac Surgery** Herbert E Warden (by invitation), Richard A DeWall (by invitation), J Bradley Aust (by invitation), Morley Cohen (by invitation), Raymond C Read (by invitation), and C Walton Lillehei, Minneapolis, Minn
- 26 **The Arterialization of Blood as It Applies to the Mechanical Heart-Lung Apparatus.** F D Dodrill and Alfred Lui (by invitation), Detroit, Mich
- 27 **The Alveolar Carbon Dioxide Tension During Intrathoracic Operations.** Thomas F Nealon (by invitation), George T Haupt (by invitation), and John H Gibbon, Jr, Philadelphia, Pa

Monday Afternoon April 25, 1955

1:00 P.M. Executive Session (Limited to Active and Senior Members)

2:00 P.M. Scientific Session REGULAR PROGRAM

Remarks by the President, Edward S. Weller, Saranac Lake, N. Y.

- 28 Lower Esophageal Web Walter F. Bugden and T. Ernest Delmonico (by invitation) Syracuse, N. Y.
- 29 Lymphoma of the Lung and Pleura Willard Van Hazel and Robert J. Jensik (by invitation) Chicago, Ill.
- 30 Chest Wall Prosthesis Bert H. Cotton, George A. Paulsen (by invitation) and Jack Dukes (by invitation) Los Angeles, Calif.
- 31 Thymic Neoplasms Donald P. Effler and Lawrence T. McCormack (by invitation) Cleveland, Ohio

6:30 P.M. Cocktail Party Chalfonte Haddon Hall Informal

Tuesday Morning April 26, 1955

9:00 A.M. Scientific Session REGULAR PROGRAM

- 32 Pulmonary Resection in Active Cavitary (Open Positive) Tuberculosis Robert H. Holland (by invitation), John W. Fell (by invitation) and Edward S. Weller, Saranac Lake, N. Y.
- 33 Simultaneous Bilateral Resection for Pulmonary Tuberculosis in Mental Patients. F. John Lewis, M. Tausig (by invitation), B. Zimmerman (by invitation) and T. F. Perry (by invitation) Minneapolis, Minn.
- 34 Blood Volume Studies in Pulmonary Tuberculosis John H. Kehne (by invitation) and Felix A. Hughes, Memphis, Tenn.
- 35 Surgical Treatment in Tuberculosis Complicated With Pulmonary Emphysema Robert W. Newman, Perry M. Huglin, Charles I. Butler and Medford C. Towne (all by invitation) Knoxville, Tenn.
- 36 Surgery for Cavitary Tuberculosis in Patients With a Single Lung Francis M. Woods and Norman J. Wilson, Boston, Mass.
- 37 Surgical Treatment of Pulmonary Histoplasmosis With MRD 112 as an Adjunct John W. Polk (by invitation), Chas. A. Brasher (by invitation), Joao de Castro (by invitation) and W. W. Buckingham, Kansas City, Mo.

Tuesday Afternoon April 26, 1955

2:00 P.M. Scientific Session REGULAR PROGRAM

- 38 The Development of the Pleural Partition to Prevent Overexpansion of the Lung Following Partial Lung Resection. Isaman A. Prewer III, Angel F. Bai (by invitation) and W. M. Jones (by invitation), Los Angeles, Calif.
- Operative Thoracic Ductography Report of Three Cases Illustrating Its Value Allan S. Smith, Ralph D. Allen and Harvey W. Kausel (by invitation), Atlanta, Ga.
- 39 Complications of Mitral Valve Surgery With Particular Reference to the Post-Cardiotomy Syndrome Charles B. Lipstein, New York, N. Y.
- 40 The Surgical Treatment of Aortic Stenosis C. P. Bailey, H. F. Bolton (by invitation), W. I. Jones (by invitation) and H. T. Nichols (by invitation), Philadelphia, Pa.

- 42 **The Use of Shunts in the Resection of the Thoracic Aorta** J Maxwell Chamberlain, Robert Klopstock, Peter Parnassa (by invitation), and A Grant (by invitation), New York, N Y
- 43 **Patent Ductus Arteriosus With Hypertension Analysis of Cases in Which Operation Was Performed.** F Henry Ellis (by invitation), John W Kirklin, and Earl H Wood (by invitation), Rochester, Minn

NOTICE

Those who are on the program are reminded that by action of the Council all papers are to be handed to the Recorder *immediately* after their presentation. Any paper which is not thus submitted immediately runs a risk of not being published.

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Original Communications

BRONCHOSPIROMETRIC STUDIES DURING THORACIC SURGERY

FRANK F. MARTIN, M.D., FRANK MACDONALD, M.D., AND WILLIAM W. STEAD, M.D.
MINNEAPOLIS, MINN.

INTRODUCTION

PREVIOUS studies from this laboratory have confirmed the frequent development of severe acidosis during intrathoracic surgery performed under general anesthesia.^{1,2} This acidosis is respiratory in nature due to inadequate alveolar ventilation despite the maintenance of a minute volume of ventilation in excess of that found preoperatively. No objective information, however, has been available on the relative contributions of the two lungs during controlled respiration in thoracic surgery. Through an adaptation of bronchospirometric techniques it was possible to measure ventilation and also carbon dioxide output of each lung throughout thoracic surgical procedures.

METHODS AND MATERIALS

The Carlens bronchospirometric catheter was used in place of the usual endotracheal tube in order to separate the ventilatory flow of the two lungs.³ The catheter was inserted immediately following induction of anesthesia. The proper placing of the catheter was easily checked by auscultation during ventilation of each lung separately. Auscultation over the anterior upper left chest area assured that the catheter had not been inserted beyond the orifice of the left upper lobe bronchus. The pilot balloons served as a constant check on the adequate inflation of the tracheal and bronchial balloons thus assuring against leaks.

The general principles and techniques of measuring ventilation during surgery have been described previously.¹ Fig. 1 illustrates the method used to record total ventilation and to obtain expired air samples. The expired air from each side was measured by separate volume displacement gas meters, the accuracy of which was determined to be within 1 per cent. Two liter mixing chambers were inserted in the expiratory arm of each circuit. A sparkless electric fan in each chamber assured adequate mixing of gas samples.

1. F. F. Martin, F. MacDonald, and W. W. Stead, *Artificial Ventilation in the Thorax*, 1953, University of Minnesota Press.
2. F. F. Martin, F. MacDonald, and W. W. Stead, *Artificial Ventilation in the Thorax*, 1953, University of Minnesota Press.
3. F. F. Martin, F. MacDonald, and W. W. Stead, *Artificial Ventilation in the Thorax*, 1953, University of Minnesota Press.

The expired air from each circuit was returned to the anesthesia machine after passing through the mixing chamber and volume meter. Ventilation was recorded at five-minute intervals. Expired air samples were collected every thirty or sixty minutes. Arterial blood samples were obtained through a sterile polyethylene tube placed in the brachial artery. These were drawn

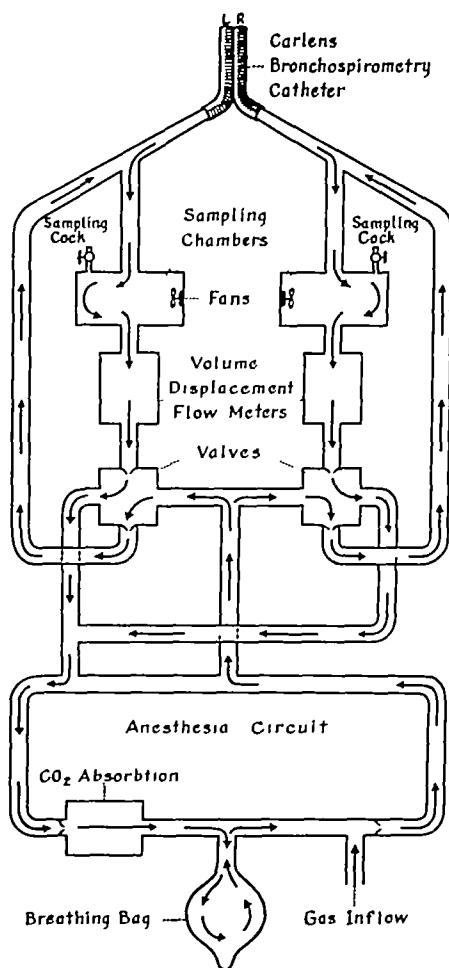


Fig 1—Diagram of anesthesia and metering sampling circuits used

simultaneously with the collection of expired air samples. The carbon dioxide concentration in the expired air was determined by standard Haldane techniques. A pH determination of whole blood was made in a glass electrode using a Cambridge potentiometer as described by Wilson.⁴ Arterial blood carbon dioxide content was measured by the method of Van Slyke and Sendroy.⁵ Carbon dioxide tension of arterial blood was calculated from the pH and carbon dioxide content of arterial blood using the nomogram pre-

pared by Singer and Hastings.⁶ Alveolar and dead space ventilation for both lungs together can be determined from the above information as described previously.⁷

Ten patients (24 to 61 years of age) were studied during intrathoracic surgery. Table I lists the diagnosis, the degree of pulmonary resection and an estimation of the disease in the contralateral lung. The extent of pulmonary resection varied from only a biopsy to lobectomy. Anesthesia consisted of intravenous Pentothal sodium and curare supplemented with nitrous oxide. Manually controlled positive pressure respiration was practiced throughout the operative period. All of the patients except one were operated upon in the semilateral thoracotomy position. H. W. was operated on in a full lateral position. The values indicated as prethoracotomy were obtained after the patient had been positioned on the operating table but before the chest had been opened.

TABLE I. CLINICAL DATA

PATIENT	AGE	SURGICAL PROCEDURE	DIAGNOSIS	CONTRALATERAL DISEASE
W. S.	24	RUL nodulectomy	Pulmonary tuberculosis	Left thoracotomy with subsegmental resection and wedges LUL done 2 months previously
H. C.	46	Subsegmental RUL	Pulmonary tuberculosis	None
G. F.	20	LLL lobectomy	Pulmonary tuberculosis	Minimal fibrosis, left apex
J. B.	20	Resection of segments left 7, 8, 9, 4, 5	Bronchiectasis	None
H. W.	51	Exploration and biopsy (not resectable) left	Bronchiogenic carcinoma	Multiple small calcifications throughout both lung fields
A. S.	26	RUL lobectomy	Pulmonary tuberculosis	Fibronodular infiltrate, left 2nd interspace
K. N.	20	Subsegmental LUL. Segmental B.	Pulmonary tuberculosis	Subsegmental resection and nodulectomies, right
D. S.	32	Subsegmental nodulectomy PUI	Pulmonary tuberculosis	Dense infiltrate left apex
A. G.	61	Two subsegmental resections and nodulectomy RUL	Pulmonary tuberculosis	None
C. G.	47	Segmental 1, wedge 2 RUL	Pulmonary tuberculosis	Fibronodular infiltrate, left apex

LLL = Left lower lobe

UL = Left upper lobe

RESULTS

Table II A gives the values for total ventilation, per cent of total ventilation contributed by the operated lung, total carbon dioxide excretion and per cent of carbon dioxide output by the operated lung prior to and at varying time intervals following opening of the chest in each of the ten patients. Table II B and C gives the arterial pH, arterial carbon dioxide tension, calculated alveolar ventilation and dead space ventilation values found in four patients.

Fig. 2 presents the mean values for total ventilation, per cent of ventilation contributed by the operated lung and per cent of carbon dioxide excretion by the operated lung prior to and at time intervals following opening of the chest.

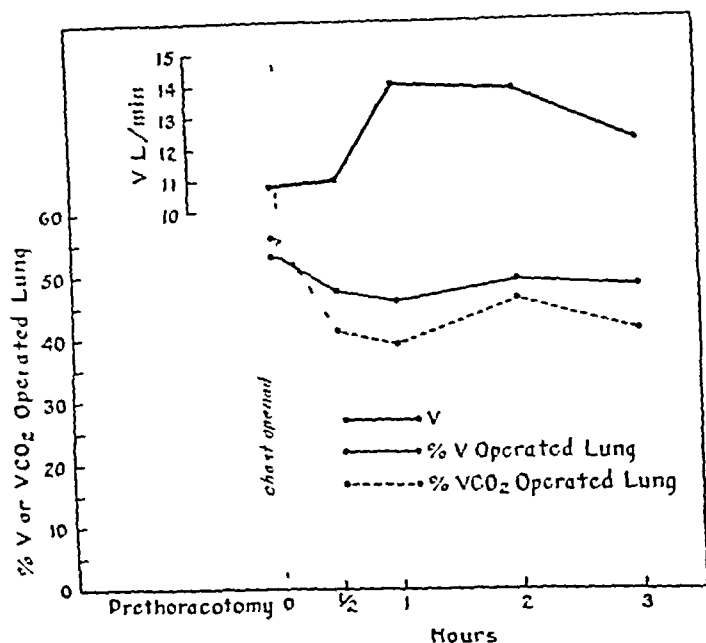


Fig. 2—Mean values found in ten patients for total ventilation (V) per cent of total ventilation by the operated lung ($\% V$) and per cent of total carbon dioxide excretion by the operated lung ($\% VCO_2$) prior to and at time intervals following opening of the chest.

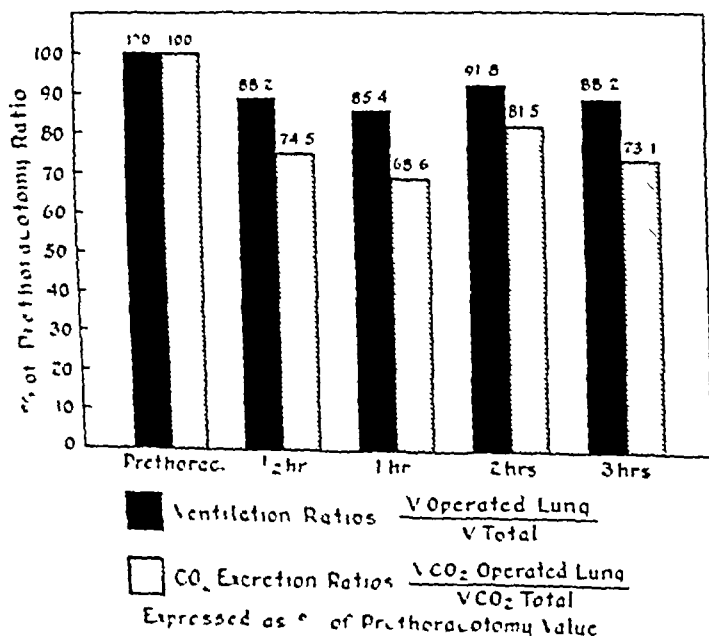


Fig. 3—Ventilation (V) and carbon dioxide excretion (VCO_2) ratios expressed in per cent of prethoracotomy values at time intervals following the opening of the chest.

Fig 3 gives the mean ventilation and carbon dioxide excretion ratios expressed in per cent of prethoracotomy values at time intervals following opening of the chest

A decrease in the proportion of ventilation and of carbon dioxide excretion by the operated lung occurred in all ten patients following opening of the chest. This decrease persisted throughout the procedure.

The decrease in the proportion of carbon dioxide excretion by the operated lung following the opening of the thorax was consistently greater than the decrease in the ventilation ratio during this period (Table II, A and Figs 2 and 3).

Some of the patients had total carbon dioxide excretion values, following opening of the chest, considerably greater than that found prior to thoracotomy. This rise was not noted in earlier studies.¹

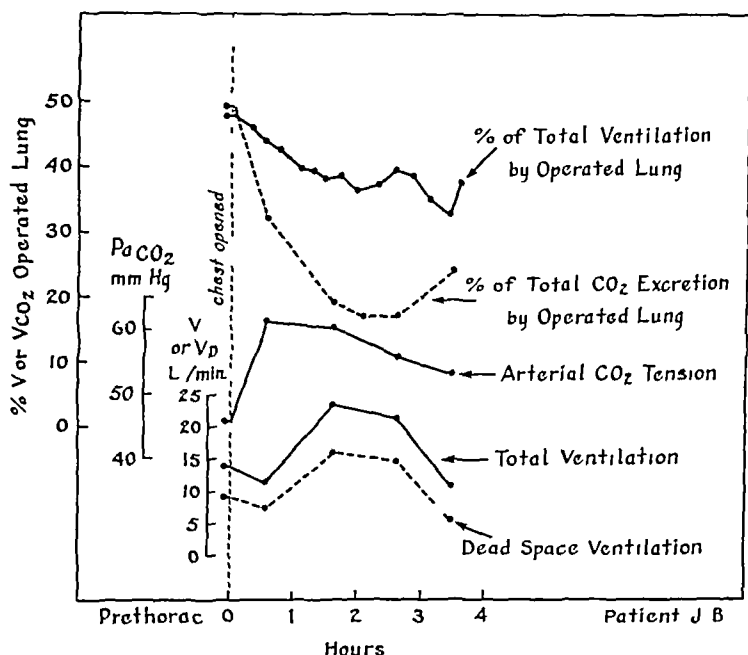


Fig 4—Values of per cent of total ventilation contributed by the operated lung, per cent of carbon dioxide excretion by the operated lung, arterial carbon dioxide tension, total ventilation and dead space ventilation at time intervals in patient J B. Resection of basal segments and lingula was performed for bronchiectasis.

The four patients in whom blood studies were made revealed a usual increase in dead space ventilation following opening of the chest. The decrease in alveolar ventilation following opening of the chest was not as consistent as was found previously.¹

Patient J B (Fig 4) showed a moderate fall in proportion of ventilation contributed by the operated lung following the opening of the chest which was accompanied by a marked fall in the proportion of carbon dioxide excretion

by that lung. The alveolar ventilation was not increased sufficiently to maintain a normal arterial carbon dioxide tension in the presence of an increased production of carbon dioxide even though the total ventilation increased up to 23.5 liters per minute. Most of the ventilation at these relatively high minute volumes consisted of dead space ventilation.

DISCUSSION

The changes noted in ventilation must be considered in the light of the anesthetic techniques applied. The combination of Pentothal sodium and curare virtually eliminates any ventilation by the patient himself. Therefore, in our patients ventilation was produced by means of positive pressure inspiration and passive expiration. Our data cannot be applied to systems employing unassisted or partially assisted respiration.

The decrease in proportion of ventilation performed by the operated lung during thoracotomy could be due to a decrease in ventilation of the operated lung, an increase in ventilation of the unoperated lung, or both. A decrease in ventilation of the operated lung may in part be related to manipulation and limitation in expansion of the lung by the surgeon. Areas of atelectasis and partial or complete bronchial obstruction could change the physical characteristics of the lung enough to affect ventilation. Clamping and resection of pulmonary tissue must play a role although patient H. W., in whom only a small biopsy was done, demonstrates that this cannot be the only factor since he showed changes similar to those patients undergoing more extensive pulmonary resection. Increased ventilation of the unoperated lung could occur if the characteristics of the closed chest side were altered by mediastinal mobility and by curarization of the chest wall muscles to permit greater ventilation with the same level of applied pressure.

The only data presented of individual lung function are ventilation and carbon dioxide excretion. More important physiologic measurements of the individual lungs would be alveolar ventilation and dead space ventilation. The latter simply represents the difference between alveolar ventilation and total ventilation. Accurate alveolar carbon dioxide tensions must be known, however, to determine the alveolar ventilation of a lung. The determination of these more important physiologic measurements must await the development of newer techniques applicable during surgery. Our data do not allow estimation of alveolar ventilation of the individual lung. The proportion of alveolar ventilation assumed by the operated lung would fall to a greater extent than the fall in ventilation ratio if the dead space ventilation of each lung remained the same, changed proportionately, or if the dead space in the operated lung increased more than in the unoperated lung. Only if there were a greater increase of dead space ventilation by the contralateral lung than by the operated lung could there be an equal or lesser fall of the alveolar ventilation ratio than of the total ventilation ratio. It is highly improbable, however, that the latter would occur.

Carbon dioxide output of each lung is a complex function of alveolar ventilation, blood flow, venous carbon dioxide tension, and inspired carbon

dioxide tension Alveolar ventilation is relatively the most important factor determining carbon dioxide excretion within usual physiologic ranges The finding of a greater decrease in carbon dioxide output ratios than in ventilation ratios is consistent with the described greater relative decrease in alveolar ventilation Variations in dead space ventilation and in pulmonary blood flow may well have occurred in one or both lungs, but one need not assume changes in these factors to explain the alteration observed in carbon dioxide output ratios

The application of the presented data to clinical situations must be made with caution It is clear, however, that the contralateral lung must assume a greater proportion of respiration during thoracotomy done under Pentothal sodium curare anesthesia and manual positive pressure ventilation than it does prior to surgery It follows that the function of the unoperated lung must be sufficient to meet this increased demand, or accumulation of carbon dioxide will occur In most cases the operated lung did contribute substantially to ventilation and carbon dioxide output

SUMMARY AND CONCLUSIONS

1 Differential lung ventilation and carbon dioxide excretion have been measured in ten patients undergoing resectional pulmonary surgery performed under Pentothal sodium curare anesthesia with manually controlled positive pressure respiration Four of the patients had concomitant determinations of arterial pH, arterial carbon dioxide content and calculation of arterial carbon dioxide tension, total alveolar ventilation and total dead space ventilation

2 A consistent decrease was present in the proportions of ventilation and of carbon dioxide excretion performed by the operated lung during thoracotomy

3 The proportion of the carbon dioxide output performed by the operated lung consistently fell to a greater extent during thoracotomy than did the proportion of ventilation

We are indebted to Shirley Lundgren and John Oganovich for technical assistance

REFERENCES

- 1 Stead, W W, Martin, F E, and Jensen, N K. Physiologic Studies Following Thoracic Surgery IV The Mechanism of the Development of Acidosis During Anesthesia, *J THORACIC SURG* 25 435, 1953
- 2 Martin, F E, Stead, W W. Physiologic Studies Following Thoracic Surgery III Ventilatory Studies in the Immediate Postoperative Period, *J THORACIC SURG* 25 417, 1953
- 3 Carlens, E. A New Flexible Double Lumen Catheter for Bronchspirometry, *J THORACIC SURG* 18 742-746, 1949
- 4 Wilson, R H. pH of Whole Arterial Blood, *J Lab & Clin Med* 37 129-132, 1951
- 5 Van Slyke, D D, Sendroy, J, Jr, and Liu, S H. Manometric Analysis of Gas Mixtures I. The Determination, by Simple Absorption, of Carbon Dioxide, Oxygen, and Nitrogen in Mixtures of These Gases, *J Biol Chem* 95 509, 1932
- 6 Singer, R B, and Hastings, A B. An Improved Clinical Method for the Estimation of Distribution of Acid base Balance of Human Blood, *Medicine* 27 223-242, 1948
- 7 Stead W W and Soucheray, P H. Physiologic Studies Following Thoracic Surgery I Immediate Effects of Thoracoplasty, *J THORACIC SURG* 23 453, 1953

RESECTION FAILURES IN PULMONARY TUBERCULOSIS

RALPH J SCHLOSSER, M.D.,* AND FRED J JARVIS, M.D.**

SEATTLE, WASH

PULMONARY resection has assumed a major role in the present treatment of tuberculosis. The indications enunciated by early pioneer centers¹⁻⁵ were quite readily accepted. These included failure of thoracoplasty and pneumothorax treatment, unilateral destroyed lung, bronchostenosis, empyema with parenchymal disease, tuberculoma, and blocked cavity. The addition of more effective antibiotic therapy, however, and a more aggressive attitude toward caseous residua have increased differences of opinion regarding indications. As a background for analysis of the present resection program employing current concepts of therapy, a study of the reasons for failure of a surgical group prior to the use of long-term drugs was undertaken. This study embraces the period from June 4, 1946, through Sept 17, 1951, during which time 400 patients had resections for pulmonary tuberculosis. It thereby affords observation of all patients for a period longer than two years. Failure of resection has resulted in early death (surgical mortality), late death (after six weeks) readmission to hospital due to exacerbation or reactivation, multiple resection, and other failures because of continuation of positive sputum or changing x-ray. An outline of the resection program and a summary of end results as derived from follow-up examination also are presented.

The history of the resection program is graphically illustrated by Figs 1 and 2. Beginning in June, 1946, major resections were used in increasing incidence, reaching a peak in 1950, declining in 1951 and 1952, to regain new impetus during 1953. The initial trend culminating in the first "high" in 1950 reflects the changing policy in the indication for pulmonary resection versus thoracoplasty. The second peak in 1953 follows the advent of long-term antibiotic therapy and reflects the increasing incidence of segmental and localized resection.

Within the period of this study, streptomycin and para-aminosalicylic acid came into general use. The first 38 patients were operated upon without benefit of these drugs. The remaining 362 all received antibiotics. Usually 1 Gm. of streptomycin was given daily from one to fourteen days before surgery and from two to six weeks thereafter. A few patients with acute and very extensive disease were treated more vigorously and for a longer time. Prior to surgery the average patient was treated with a conservative and

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prolonged period of bed rest. Following resection (except for brief post operative ambulation) bed rest prevailed for a minimum of six months, followed by graduated exercise. This program usually resulted in discharge of the patient about one year after surgery.

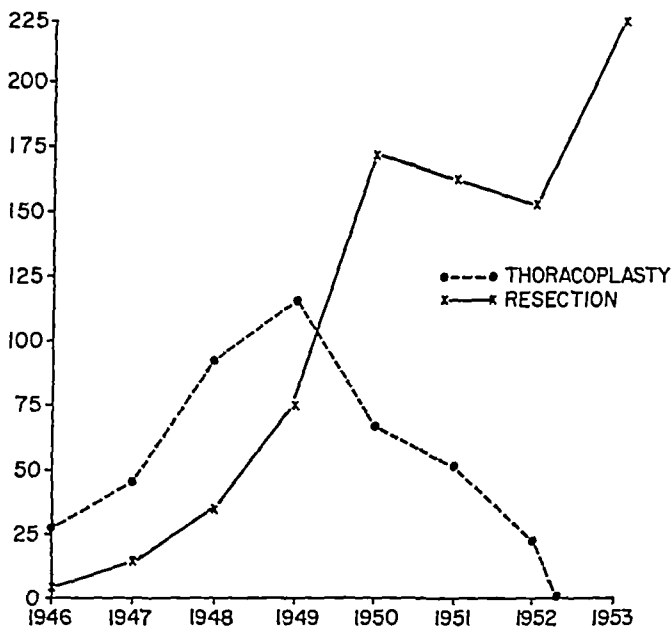


Fig 1—Annual incidence of thoracoplasty and resection

In general the character of the lesions in cases coming to surgery after therapy with bed rest and short-term drugs has differed widely from that seen at the present time with use of long-term drugs. Greater problems were encountered technically. In some cases the finding of unsuspected disease in a lung resulted in pneumonectomy rather than lobectomy. In other instances, pneumonectomy was forced due to vascular difficulties during surgery. In many cases, for fear of reactivation, masses of functioning lung were removed in order to eliminate scattered foci of caseous disease. At present these lesions would be resected locally with minimal sacrifice of functioning lung, or long-term drugs would be used to complete their healing.

SELECTION OF PATIENTS

Patients are selected for surgery by a majority vote of the Staff, meeting weekly in general conference. The pertinent data and x-rays are presented by the physician in immediate charge of the patient, full discussion ensues, and opinions are expressed before a vote is taken. For the indication and the type of surgery to be done, laminographic x-ray study has been employed extensively and reliably. Similarly, with the inauguration in 1948 of facilities for the adequate study of pulmonary function, decisions for or against surgery have been influenced by the findings of the pulmonary physiologists.

TYPE OF SURGERY

During the period of this analysis, there were 411 resection procedures on 400 patients as performed by the three consultant thoracic surgeons of Firland Sanatorium* and by the surgical residents under their supervision. The resection procedures include 160 pneumonectomies, 244 lobectomies, six segmental resections, and one nodulectomy. A high percentage of the pneumonectomies and lobectomies followed unsuccessful thoracoplasty procedures. Segmental resection was not done extensively, a reflection of the opinion that removal of all significant areas of caseation by more extensive resection protected the patient against reactivation, whereas cutting through disease was dangerous. Thus, at times, considerable functional lung tissue was sacrificed.

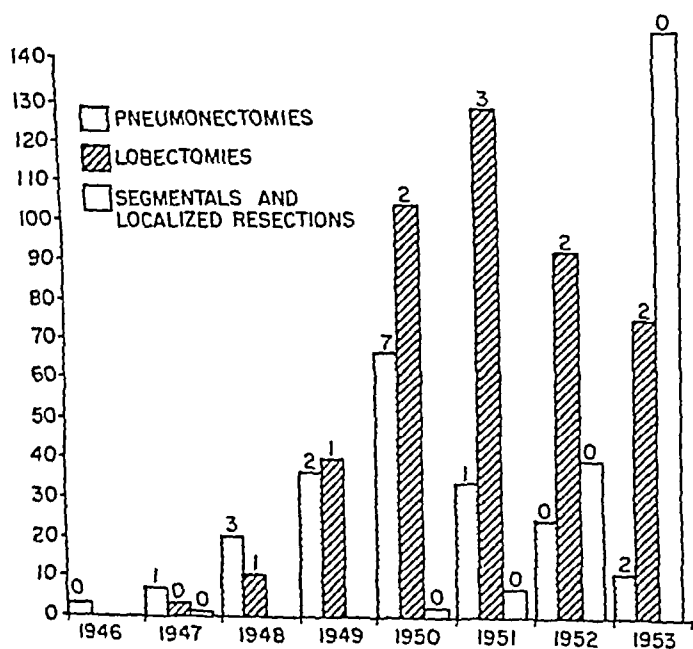


Fig. Types of resection. The number of surgical deaths for each type and year is indicated by the number above each column.

SURGICAL MORTALITY

Twenty-one patients died within six weeks of surgery, an arbitrary interval accepted by most surgeons in computing surgical mortality. In addition, some of our late deaths were directly accountable to surgical complications. The over-all surgical mortality of the 411 patients was 5.1 per cent. Fourteen deaths occurred following pneumonectomy, a mortality rate of 8.7 per cent. The relatively large number of pneumonectomies, in itself indicates the severity of the disease process and the greater likelihood of in-

* Firland Sanatorium, MD, Edward D. Latham, MD, and Walter O. Miller, MD.

volvement of other systems or the opposite lung. Seven deaths followed lobectomies, a mortality of 2.9 per cent. There were no deaths in the segmental or localized resections.*

Examination of the protocols of the patients dying in the early postoperative period makes it apparent that a most important factor accounting for failure was inadequate functional reserve. It is emphasized that the functional insufficiency of these patients was observed clinically by studying their behavior during and following surgery. Complications in the nature of retained secretions, paradoxical respiration, delayed expansion through temporary surface leakage of air, phrenic and recurrent laryngeal nerve paralysis, successfully weathered by patients with greater reserve, have in these patients been fatal. Three patients in this group presented insurmountable respiratory difficulties under anesthesia. This resulted in prolonged anoxia with respiratory failure in the immediate postoperative period.

Major surgical accidents accounted for four deaths. Two of these occurred during extrapleural pneumonectomy, in one an infected oleothorax and bronchopleural fistula, and in the second a two-stage unsuccessful thoracoplasty complicated by empyema, were surgical exercises of major magnitude. A third patient died on the operating table during closure of the skin when the ligature on the pulmonary artery gave way. The fourth died of hemorrhage from the chest wall following thoracoplasty three weeks after pneumonectomy. Major blowout of a bronchial stump has been responsible for early death only twice. Pulmonary embolism causing death was proved in two cases, suspected in two others.

LATE DEATHS

The number of patients dying six weeks or later postoperatively was greater than the number succumbing in the early postoperative period. 33 late, 21 early. These complications for the most part are directly related to the surgery, although 11 patients succumbed during the second, 7 during the third, and 2 during the fourth year. Empyema with bronchopleural fistula accounted for a third of these late deaths. Two patients had difficult extrapleural pneumonectomies with areas of caseation encountered during surgery, a factor frequently resulting in empyema. In one patient an empyema was demonstrated by needle aspiration eight months after surgery. She died eight days later from disturbance of cardiac conduction mechanism, probably ventricular fibrillation. Autopsy revealed no fistula. The remaining seven patients were found to have a coexistent empyema and bronchopleural fistula.

Functional inadequacy finds its late expression in cardiac death. Nine patients, including two listed by the coroner without autopsy as "coronary occlusion and sclerosis" and "acute cardiac insufficiency, chronic valvular heart disease" died late cardiac deaths. It seems probable that this group

*Two hundred and sixteen localized resections have been performed prior to April 1, 1954 with no deaths.

developed cardiac strain due to inadequate pulmonary functional reserve. Other late deaths include five patients who expired from progressive disease. As an indication of the severity of the disease four required pneumonectomy. Nine other patients died of indirect or unrelated causes.

READMISSIONS

Of primary interest are the 27 patients who had undergone resectional therapy and left the sanatorium only to be readmitted for further treatment. Eighteen of these patients had been discharged with Staff approval. Two were given disciplinary discharges and the remaining seven left the hospital against medical advice. Evidence of reactivation of disease was demonstrated either by change in x-ray shadow or by the discovery of positive sputum or both. Fifteen (58 per cent) returned during the first two years after surgery, the greatest number reactivating during the second year. However, a significant number returned in the fourth, fifth and sixth years (Table I). This contrasts with the series of segmental resections reported by Chamberlain and associates⁶ in which late reactivations have not represented a serious problem. Two thirds of the exacerbations in their series occurred within the first six months.

TABLE I TIME OF POSTSURGICAL READMISSION

NO. OF PATIENTS		
First year	3	} 58%
Second year	12	
Third year	5	
Fourth year	4	
Fifth year	2	
Sixth year	1	
Total	27	

Twelve of the readmitted cases had undergone pneumonectomy. This being a significantly high number relative to the lobectomy cases, it would seem again to reflect the greater severity of the original disease and probably the lesser resistance of the host. Of the 15 postlobectomy patients, the exacerbation occurred in the contralateral lung in six, in the operated lung in five, and in both lungs in two. Two patients showed no x-ray change and were readmitted for investigation of the source of a positive sputum culture. In one, an ulceration of the stump was found. In the other, no source was determined. Both cleared with additional antibiotics. The efficacy of drug therapy is attested by the fact that it was possible to convert the sputum and ultimately discharge 16 of these patients on long term antibiotics. Six of the readmission group remain in patients but with a favorable outlook. One patient who had undergone pneumonectomy was recently readmitted with cavity disease. Prognosis is guarded. Two remain outside the sanatorium with intermittently positive cultures without known source. One has died of unrelated causes, and one is not available for follow up.

MULTIPLE RESECTIONS

Eighteen* of the series of 400 patients have undergone multiple resections, three as planned in their over-all therapy. Of the remaining 15 patients, 13 required a second resection because of recrudescence of disease postoperatively, one because of empyema, and one because of stump blowout. Increased disease, as seen by x-ray and by sputum studies, occurred from two to fifteen months following the initial surgery, with the average interval being eight months. We noted the extension of disease in the operated lung in ten of the thirteen cases, in both the operated lung and the contralateral lung in two cases, and in the contralateral lung alone in one instance. All of these patients received some form of short-term antibiotics postoperatively, consisting of daily streptomycin for a period of two to four weeks, some receiving PAS as well. Twelve patients were treated with streptomycin preoperatively for a short period.

Twelve of the eighteen patients ultimately achieved a good result following removal of more lung tissue. At the present time they are living and well with stable x-rays and negative sputum. Two remain as inpatients, one still has a positive sputum and a guarded prognosis under long-term drug therapy, the other is classed as chronic custodial. Two patients were discharged with advice, with negative sputum, but are lost to follow-up. Two are dead, one appearing with the early deaths and the second with the late deaths.

OTHER RESECTION FAILURES

Because of positive sputum or changing x-ray, 15 additional patients are also classified as failures. Five remain in the hospital. They all have experienced extension of disease postoperatively and persist with positive sputum despite prolonged drug therapy. In four of the five, inadequate functional reserve has prohibited further surgical consideration. In fact, a study of their postoperative course suggests that borderline function complicated by sputum retention and paradoxical respirations probably played a large part in promoting the extension of their disease. In one, repeated exacerbation of sickle cell anemia seemed directly related to the extension of her tuberculosis. Four other patients are at home. One has a low functional reserve with intermittently positive sputum, another has persistently positive sputum cultures without known source. A third is awaiting recheck studies, after an isolated positive sputum culture, and questionable x-ray changes. The fourth is being sought for readmission following the development of positive sputum and definite x-ray changes seven years after pneumonectomy. Five other patients with late isolated positive cultures and without x-ray changes are under observation. One patient with negative sputum is awaiting recheck after questionable x-ray change in the opposite lung.

*Eleven patients were operated upon a second time during the period of this study, thus accounting for the 411 resection procedures on 400 patients. The remaining seven had their second procedure subsequent to this study.

†Including the two of the multiple resection group the persistent inpatients total seven.

SUMMARY OF RESULTS

We have been able to follow 357 of the 400 patients, 89 per cent. Table II shows the present status of the patients followed.* Seventy-eight per cent ultimately achieved a good result and are at home, sputum negative. The total death rate of the patients followed was 15 per cent, the surgical mortality 5.1 per cent. The results of other series as cited by Shabart and Samson,⁷ to which we have added our own results for comparison, are shown in Table III.

TABLE II PRESENT STATUS OF 357 PATIENTS FOLLOWED

<i>At Home</i>	Sputum negative	279	(78%)
<i>At Home</i>	Sputum positive	10	
	Isolated positive culture	5	
	Condition unsatisfactory (MHB)	4	
	? changing x ray	1	
<i>In Sanatorium</i>	Readmitted	7	14
	Persistent inpatients	7	
<i>Dead</i>	Early surgical deaths	21	54
	Late deaths	33	

TABLE III RESULTS OF PULMONARY RESECTION FOR TUBERCULOSIS

AUTHORS	NO	WELL	SICK OR COMPLICATIONS	OPERATIVE DEATHS	TOTAL DEATHS
Gale Sweet Day, Chamberlain, Bailey, Himmelstein, Brantigan (per cent)	745	496	95	47	177
Glover (per cent)	347	665	126	63	183
Davidson (per cent)	200	70	77		22
Overholt (per cent)	426	151	45		20
Shabart Samson (per cent)	95	755		38	87
Schlosser, Jarvis Operations	411	709	13	89	204
Patients followed (per cent)	357	65	14	6	17
		68	14	6	18
		279	24	21	54
		75	7	51	15

DISCUSSION

In analyzing our resection failures, we have not considered the morbidity of the operation. Small empyemas, which disappeared spontaneously after aspiration or required small thoracoplastics, wound infections, stump ulceration and the other postoperative annoyances familiar to the thoracic surgeon were all troublesome when present. In some instances they delayed a patient's discharge from the hospital although his disease remained under control. Certain patients with mild respiratory embarrassment prior to surgery achieved stability of disease following resection and now remain with relatively the same extent of pulmonary crippling.

No effort has been made to determine the number of patients whose entire lung was removed as planned in their therapy, compared to the number

* We are indebted to Mr. Percy Clark, M.D., and Mr. Clyde Nesbitt, M.D., for their assistance in obtaining our follow-up data and to Dr. John T. B. for his assistance in the Division of Tuberculosis, County Health Department.

of pneumonectomies performed because of vascular difficulties encountered technically or from the finding of unsuspected necrotic disease in the second lobe. Increased use of laminographic x-ray, as experience was gained, resulted in fewer unsuspected lesions. In future studies we hope to correlate closely the disease as seen by standard films and laminograms with that recovered in the surgical specimen.

At the outset of this study we were interested particularly in the deaths and complications related to inadequate pulmonary function. It is apparent to us from examination of such failures that the postoperative management of borderline function cases is vitally important. Had we been able to prevent or minimize the paradoxical respirations, retained secretions, minor peripheral lung leaks, phrenic or recurrent laryngeal nerve paralyses that occurred, at least during the immediate postoperative period, certain of these patients might have survived. In others, early tracheotomy, as suggested by Carter and Giuseffi⁸ has been invaluable in their postoperative management. Other patients were felt to have adequate function and yet have already died with cor pulmonale. A major question pertains to the future course of those patients with low function reserve following resectional therapy. Is cor pulmonale eventually their fate? From our experience it would seem that this will be verified in a significant percentage.

Stump ulceration was a common but not a formidable problem. The late Dr. Lewis R. Roll⁹ of the Fuland Staff made a detailed study of the first 389 of these 400 patients and reported an incidence of over 24 per cent tuberculous ulceration of the bronchial stump. He predicted that stump ulceration would be a "minor problem in the future" once long-term drug therapy was prescribed with full knowledge of the sensitivity of the organisms to the drugs. Stump ulceration is now an extremely uncommon occurrence in the large group of patients subsequent to this series who have undergone segmental resection.

Early opening of a bronchial stump, indicating its insecure closure, has been uncommon. Even following pneumonectomy, fistula through the closed bronchial stump has usually been late in developing. In reoperated cases, search for the source of the bronchial fistula following lobectomy or segmental resection has shown the leak to arise from smaller bronchi in the raw surface of the lung. To us, these are indications that most of the late bronchial complications stem from unrecognized accumulations of fluid within the pleura, as pointed out by Stemmermann, Daniels, and Auerbach.¹⁰ Contamination and low-grade infection in such accumulations, although frequently small, may result in a pleural abscess in which tubercle bacilli or pyogens of low-grade virulence are present. Such empyemas, we feel, always tend to seek an exit to the outside. They insinuate themselves through planes of low resistance, such as a tenuous closure of a bronchus, or through a small bronchus on lung surface formerly closed only by fibrin. The result is late bronchial fistula. To reduce these hazards to a minimum, it is necessary to give meticulous care to the obliteration of all pleural space as early as possible in the postoperative period.

In studying the readmission patients it is apparent that the term "spread" is an extremely inaccurate one to apply to the manner of exacerbation of disease. In most instances there has been prior to surgery a definite tuberculous lesion in the region of the so-called "spread." Even though no shadow is seen in the x-ray it is logical to accept the existence of a small necrotic lesion which later reactivates. The limitations of the ordinary x-ray film in demonstrating minute but significant lesions are recognized. Moreover since "spread" connotes a reinoculation of lung parenchyma through a bronchogenic source, if this source is eliminated by a resection, as indicated by sputum conversion, then reactivation would seem to be the mechanism of subsequent extension of disease.

SUMMARY AND CONCLUSIONS

1. An analysis of the failures in a series of 400 consecutive patients undergoing resection for pulmonary tuberculosis is presented. Detailed study includes the early and late deaths, the readmission and multiple resection groups and other failures. We feel that this study is helpful in the evaluation of present trends in surgical therapy of pulmonary tuberculosis.

2. Inadequate functional reserve has been a major cause of both early and late death.

3. Whereas major blowout of a bronchial stump indicating insecure closure was responsible for early death in two cases, a third of our late deaths were accountable to empyema with late bronchopleural fistula. The etiology of these late hazards in most cases is felt to stem from seemingly innocuous but persistent pleural fluid.

4. As demonstrated by our readmission and multiple resection group, residual caseous disease is a definite threat to a patient's future. In certain of these patients somewhat greater extension of the initial resection might well have prevented later surgery.

5. Reactivation rather than "spread" is usually the manner of exacerbation of disease following resectional therapy.

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LATE RECONSTRUCTION OF CONGENITAL ESOPHAGEAL ATRESIA BY INTRATHORACIC COLON TRANSPLANTATION

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THE optimal repair of congenital tracheo esophageal fistula of the infant at present consists of ligation of the fistula and establishment of alimentary canal continuity by primary end-to-end esophageal anastomosis¹ In some instances the primary suture of proximal esophageal pouch to distal esophagus is difficult or impossible because of the great distance between the esophageal ends This situation has generally been managed by one of three methods (1) mobilization of the entire distal esophagus and upper stomach to allow end-to-end esophageal anastomosis, (2) gastric mobilization and intrathoracic gastric transplantation to permit esophagogastrostomy, and (3) delay of reconstruction by performance of cervical esophagostomy and abdominal gastrostomy The first two alternatives permit primary reconstruction but entail either an anastomosis of proximal esophageal pouch to a widely devascularized distal esophagus, or to the stomach which, by virtue of its presence within the thorax, imposes the disadvantages of gastric distention and regurgitation The latter method of creation of skin stomas has necessitated prolonged and complicated later reconstructive procedures

Two years ago we operated upon two infants for tracheoesophageal fistula, and primary anastomosis was not done In the first instance, the anastomosis was done but ended with an obviously dangerous suture line Rather than accept the almost certain leak and possible death, it was decided to perform cervical esophagostomy and abdominal gastrostomy until later reconstruction could be done In the second case, there was complete atresia without a fistula A two-inch fibrous cord connected the proximal and distal esophageal segments Cervical esophagostomy and gastrostomy seemed preferable to mobilizing the stomach

During the more than two years which intervened between the initial operations and final reconstruction, various possible reconstruction plans were considered, including the transplantation of stomach and jejunum into the chest The experiences of others with the reconstruction problem were reviewed

There appeared to be universal dissatisfaction with skin tubes used to bridge distances between the upper esophagus and stomach, chiefly because of the necessary multiple stages, the common occurrence of fistulas at either end,

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the development of strictures due to fibrosis and the extensive scarring of the anterior chest. Subcutaneous tunnels seem psychologically quite disadvantageous particularly in a child.

Gastric transplantation and esophagogastrostomy have presented the problems of gastric dilatation and regurgitation, the development of peptic esophagitis of the proximal stump, and occasional difficulty in mobilizing the stomach to reach the neck.

The chief disadvantages of jejunal transplantation and anastomosis have been related to the length of the segment which could be mobilized and carried to the neck without necrosis, and the necessity for by-passing the stomach.

Recent experience here^{4*} and elsewhere^{3, 5, 6} with intrathoracic colon transplantation following esophageal carcinoma resection led to its consideration for late reconstruction in congenital esophageal atresia. We were unable to find any report of experience with this procedure for bridging the defect of esophageal atresia.

RATIONALE OF ESOPHAGEAL RECONSTRUCTION USING INTRATHORACIC COLON

Theoretical and practical considerations indicate that intrathoracic transplantation of the right colon may avoid disadvantages of other types of esophageal reconstruction for congenital atresia of the esophagus in a child.

1. A one stage reconstruction may be accomplished by laparotomy without formal thoracotomy. The right colon may be mobilized and pulled through the anterior mediastinum via a substernal tunnel dissected without actually opening the chest wall. Immediate anastomosis in the neck and to the stomach may then be performed through separate cervical and abdominal incisions. The chief disadvantages of a one stage procedure appear to lie in the dangers associated with the five different suture lines necessary to this operation, as well as the possibility of contamination of the mediastinum. The development of antibiotic regimens and better methods of preparing the colon for anastomosis probably obviate most of this. That the entire procedure can be carried out within a reasonable time is evidenced by the four-hour operating time in these cases which included time used to make photographic records.

2. The blood supply should be adequate and may be tested prior to division of any vessels by placing vascular clamps on the right colic vessels. In almost every instance the collateral circulation through the marginal arteries should be sufficient.

3. That the right colon will be long enough is attested by extensive clinical and experimental data and by experience in our autopsy room where in each instance a sufficient length of right colon could be developed to bridge the distance between the neck and the stomach.

4. The incidence of peptic ulceration should be lower than when stomach, stomach-tube, or jejunum is used.

5 The incidence of fistula formation at the site of anastomosis should be no higher than with other transplantations, particularly since this is probably related to the inadequate blood supply of the other types of anastomosis

6 The procedure does not by-pass the stomach as does the jejunal transplantation.

7 Intrathoracic reconstruction is better than subcutaneous placement for psychological reasons, if for no others

Possible disadvantages include stasis within the transplanted colon, compression of the stomach by the middle colic vessels passing over it, and stenosis at the anastomosis lines. In the two cases reported here Lipiodol x-ray examinations have indicated that the transplanted colon empties slowly. Regurgitation has occurred, however, only when oral feeding has been rapid. If compression of the duodenum or pylorus proves a real disadvantage, it should be possible to bring up the vessels and colon posterior to the stomach through the gastrohepatic ligament and thus avoid this compression. Should compression occur after anterior colon transplantation, gastrojejunostomy might be needed. The incidence of stenosis should be no greater than with other types of anastomosis. The use of fine interrupted nonabsorbable sutures is stressed.

CASE REPORTS

CASE 1—R C, SMH No 332854, was born to a 23 year old woman on Aug 27, 1951. Birth weight was 2,150 grams (4 pounds, 11 ounces), and delivery was two weeks prior to the expected date. Crowing respirations and moderate cyanosis were observed. Twenty nine hours later, because of an episode of excessive salivation and cyanosis, an unsuccessful attempt was made to pass a Levine tube into the stomach. After several hours and another unsuccessful attempt to pass the tube, Lipiodol injected via the tube confirmed the diagnosis of tracheo esophageal fistula. The proximal esophageal stump lay at the level of the second thoracic vertebra (Fig 1). Gas in the stomach and small bowel indicated the presence of a fistula. Preparations for thoracic exploration consisted of parenteral fluids, frequent nasopharyngeal aspiration, administration of penicillin, and placement of a cannula in the saphenous vein.

*Initial operation** was begun fifty one hours after birth under cyclopropane and ether anesthesia. The right chest was entered through the bed of the fourth rib. After ligation of the azygos vein and incision of the mediastinal pleura, the fistula between distal esophagus and right main bronchus was found. This was cut and the bronchus sutured with interrupted 5/0 silk. The proximal esophageal stump was located high in the mediastinum and was dissected into the cervical region. The small distal esophagus was dissected free to the diaphragm. The segments were now 2.5 to 3 cm apart. It was thought that primary anastomosis could be accomplished, and two posterior layers of interrupted 5/0 silk sutures were placed and successfully tied. The inner anterior layer of sutures then was placed. Upon attempting to tie these, the anterior sutures pulled out of the friable esophageal tissue and the posterior suture lines began to disrupt. Anastomosis without later disruption seemed impossible. Because of the poor condition of the premature baby and the torn condition of both esophageal ends the procedure was terminated by left cervical esophagostomy in anticipation of later esophageal reconstruction.

The following day a Stamm type gastrostomy was done under local anesthesia, leading the tube out of the left side of the abdomen from the anterior gastric surface. After an hour,

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normal saline gastrostomy injections were begun and the infant was tolerating full formula via gastrostomy by the fourth post-operative day. Right upper pneumonia cleared gradually and penicillin was stopped after seven days. The baby was discharged on the fifty-second hospital day weighing 3280 grams (7 pounds 2 ounces).

The infant was seen at intervals for changes in formula and for cleaning the tube. Except for mild difficulties with the skin around the two stomas development was excellent.

On Jan. 17, 1954 at the age of 2 years 5 months he was readmitted for esophageal reconstruction weighing 9.6 kg (21 pounds). Aside from the esophagostomy and gastrostomy openings he was normal (Fig. 2). Preoperative preparation consisted of Neomycin 0.25 Gm and Sulfathiazole 0.5 Gm every four hours via the gastrostomy tube, daily tap water enemas and nightly milk of magnesia administration.



tinued after a week. Gastrostomy fluids were begun the third postoperative day after several stools were passed and were gradually advanced from 0.5 per cent saline to full formula. He took water by mouth the fourth postoperative day. By the twelfth postoperative day he was completely nourished by mouth in the form of liquid and soft baby food. The child swallowed poorly since he had no previous experience with the act. Occasionally, if feedings were taken rapidly, several spoonfuls were regurgitated. This was controlled by feeding more slowly. Barium swallow (Fig 3) showed pooling of the material in the colon transplant but a follow up film after twenty-four hours showed complete absence of barium except for a few flecks in the colon. The wounds healed well, and he was discharged on the sixteenth postoperative day in excellent condition (Fig 4). A small fistula developed in the neck on the twentieth day, but was completely closed after four weeks.



Fig 2—Case 1. Patient, aged 2 years 5 months prior to esophageal reconstruction. Gastrostomy tube is in place.

Increasing difficulty in swallowing became apparent and Lipiodol swallow four months postoperatively showed complete stenosis of the cervical anastomosis between esophagus and cecum. Esophagoscopy confirmed this. The child was readmitted and on June 4, 1954, the cervical anastomosis was explored, finding a thin band of scar tissue completely closing the anastomotic stoma. The area was resected, and a new anastomosis was made between esophagus and cecum (three layers of interrupted 5/0 silk sutures).

The postoperative course was uneventful and the baby was discharged on the eleventh postoperative day, eating well in small amounts. As of Oct 4, four months postoperatively, the baby continued to do well.

CASE 2—M. W. SMH No 347881, the second child of a white 19 year old woman, was born on July 16, 1952, weighing "over six pounds." At each feeding the patient vomited, choked, and became cyanotic. On the third day she was transferred to Strong Memorial Hospital, weighing 2,700 grams. The tentative diagnosis of tracheo esophageal fistula was



FIGURE 1. Postoperative barium swallow showing anterior mediastinal position of transplant colon (lateral chest X-ray).

On July 16, 1952, a barium swallow showed atresia without visualization of a fistula. No air was seen in the stomach or bowel. Preoperative preparation consisted of elevating the foot of the bed to help ventilation, frequent oral pharyngeal suctioning, penicillin, oxygen, and hydration through nasogastric cannula.

At the time of operation on July 20, 1952, under general anesthesia there was esophageal atresia with a blind pouch. A transverse colon was anastomosed to the proximal and distal

esophagus Rather than transplant the stomach into the chest a cervical esophagostomy was done On the following day a Stamm gastrostomy was performed, and feedings were begun through this on the succeeding day The postoperative course was benign, and the patient was discharged on the eleventh postthoracotomy day, weighing 3,000 grams She failed to gain weight and did poorly The patient was followed in the clinic and finally was admitted to the hospital on June 21, 1953, weighing only 4,830 grams A complete study failed to show any definite cause for her malnutrition This included stool fat and trypsin content, vitamin A study, intravenous pyelogram, glucose tolerance, duodenal aspiration for enzymes, gastrointestinal series for enteric fistula, gastric acidity, and pepsin studies We were unable to build the patient up in the hospital, and, since she represented a poor risk for surgery, she was discharged on July 31, 1953, hoping she could be built up on a celiac diet Discharge weight was 5,180 grams, only 350 grams over her admission weight of 4,830



Fig 4—Case 1 Patient, fourteen days after operation abdominal and cervical incisions well healed gastrostomy tube still in place.

The third admission on Oct 12, 1953, showed her to be more malnourished, having lost 620 grams and weighing only 4,560 grams on admission All of the studies of the second admission were repeated Further studies included saliva analysis, oral and intravenous glucose tolerance tests, gastroscopy, Thorn tests, insulin tolerance tests, and nitrogen balance studies No marked abnormalities were noted although there were one or two borderline results With a high caloric celiac diet and careful nursing, the patient slowly gained weight An extensive series of tests relating gastric response to a wide variety of

simultaneous done over a five month period. She finally reached a weight of 7,530 grams and seemed quite robust and ready for surgery. She was prepared for surgery in the same manner as the patient in Case 1.

On March 23, 1954, esophageal reconstruction was accomplished exactly as in the first case. Operation time was four and one half hours including frequent interruptions for movies. The postoperative course was very benign except for a slight fever of from 100° to 101° F. which subsided after the venous cut down was removed on the third day. She was given a sucker on the eighth day and an oral diet was given on the eighteenth postoperative day. She had some regurgitation intermittently which she did not seem to know how to handle. After she was placed on six small feedings daily this was no longer a problem, and she slowly gained weight prior to discharge. On Aug. 27, five months postoperatively she weighed 18,750 grams and was steadily improving.

TECHNIQUE OF INTRATHORACIC COLON TRANSPLANTATION

The preoperative plan of operation was followed. The steps of the procedure are discussed and illustrated.

1 *Preparation* for operation consisted chiefly in administration of Neomycin and Sulfathalidine to prepare the lower ileum and colon for section. Penicillin was used as prophylaxis of mediastinal infection.

2 *Incision* into the abdomen with wide exposure was obtained by right paramedian rectus retracting incision from the xiphoid to well below the umbilicus (see scar in Fig. 4).

3 *Right colon mobilization* was performed by cutting the lateral ascending colon and cecal attachments and dissecting in the avascular retroperitoneal area. The hepatic flexure was similarly dissected with due care for the duodenum. This permitted the right half of the colon and the terminal ileum to be lifted out of the abdomen (Fig. 5).

4 *Collateral vascular testing* was done by placing arterial clamps on the right colic artery near its origin (Fig. 6). The blood supply of the right colon was now obtained from the intact middle colic artery (foreeeps point to this) and the arcades of the marginal artery. After ten minutes there were excellent pulsations in the marginal artery and vessels supplying the cecum and ascending colon.

5 *Appendectomy* was performed while the vascular clamp was in place. The mesoappendix was serially ligated, the appendix cut between a proximal singly tied 4-0 silk ligature and distal clamp and the appendiceal stump buried between a purse string cecal suture of 4-0 silk reinforced by interrupted 4-0 silk residual sutures.

6 *Section of the ileum* was performed between two obliquely placed clamps and the small vessels of the distal ileal mesentery and of the peritoneum attached to the ascending colon were ligated as well as the right colic artery and the ileocolic artery. The terminal ileal stump was turned in by a double layer of sutures. The colon could now be swung up onto the chest wall to a point in which point the transverse colon should be cut.

7 *Section of the transverse colon* was carried out to the left of the middle of the cecum with care to avoid injury to the marginal artery. The transverse



Fig 5—Right colon fully mobilized and lifted out of abdomen. Forceps point to right colic artery. Ileocecal valve and appendix lie at lower left and transverse colon at top.



Fig 6—Right colic artery and terminal ileal arteries temporarily clamped to test vascular supply to right colon through middle colic artery (forceps at right) and marginal artery.

mesocolon was actually not cut down at all. The gastrocolic ligament to the right of the level of section of transverse colon was cut down and its vessels tied.

8. End-to-end ileum-transverse colon anastomosis was now performed in two layers, the outer of carefully placed interrupted 4-0 silks and the inner as a continuous 4-0 chromic gut layer. The ileal and transverse colon mesenteries were approximated by interrupted 4-0 silk sutures to prevent small bowel herniation. The right colon could now easily be placed on the chest wall to illustrate its length (Fig. 7).



FIG. 7



FIG. 8

Fig. 7. Ileum on transverse colon lying on chest wall. Clamp is across transverse colon. Suture closing C-loop ileum and appendix inverted. Ileum at top near cervical anastomosis. Transverse colon lies below. Lung at bottom.

Fig. 8. An enteric clamp pulled through subcutaneous tunnel (ant. rib tr. lig. tunnel) into chest. Transverse colon suture to cecum. Ileum clamped. Cut end of transverse colon.

9. An anterior mediastinal dissection to form a substernal tunnel was performed. Through a low collar cervical incision the cervical investing fascia was opened transversely and the anterior mediastinal plane posterior to the



Fig 9

Fig 10

Fig 9—Anastomosis of proximal esophagus to cecum in neck, and of transverse colon to anterior surface of stomach in progress. Open two layer anastomoses were simultaneously performed by two teams.

Fig 10—Wounds closed. Gastrostomy tube remains in place. Upper neck wound was esophagostomy site and lower wound was anastomosis site. In the second case only one wound was made in the neck.

sternum entered with the finger. At the cephalad end of the abdominal wound the tip of the xiphoid was exposed and by combination sharp and blunt dissection a plane developed just posterior to the periosteum of the xiphoid process. Blunt finger dissection was now carried out both from below and above keeping close to the posterior sternal periosteum. A stay suture was placed in the tip of the cecum, and the cecum was pulled up through the substernal anterior mediastinal tunnel by means of this stay suture (Fig 8). In the second case this stay suture was omitted, and the cecum was pushed through the tunnel by hand. The team was divided in half and the following two steps of esophageal colon and colon-stomach anastomosis carried out simultaneously.

10 *Anastomosis of proximal esophageal pouch and cecum* was performed after mobilization of the esophagostomy and excision of the distal end. This

anastomosis was done entirely with interrupted 5-0 silk and consisted of three layers. There was no tension on the suture line at the end of the procedure. Fig. 9 illustrates both this and the following step in progress.

11 *Anastomosis of transverse colon and stomach* was performed near the lesser curvature low on the anterior surface of the stomach with an outer layer of interrupted 4-0 silk and internal continuous 4-0 chromic gut. The transverse colon now arched upward from the anterior surface of the stomach and passed between the lobes of the liver up into the anterior mediastinum. Palpation of the colon and its vascular supply through the middle colic artery showed no tension existed.

12 *Wound closure*. Wounds were inspected for accurate hemostasis and closed with interrupted 4-0 and 5-0 silk sutures (Fig. 10). The gastrostomy tube remained in place since this had been walled completely out of the field, and it had not been necessary to dissect the stomach away from the anterior abdominal wall.

13 *X-ray examination* of the chest revealed 75 per cent left pneumothorax in the first case and 15 per cent left pneumothorax in the second. This was removed by simple needle aspiration and the lung re-expanded completely at once. This pneumothorax was the result of mediastinal dissection, which, although done as delicately as possible, was "blind" and ruptured the weblike pleura. Despite the absence of physical signs, it must be suspected after this dissection.

At the conclusion of the steps outlined above the proximal alimentary canal of the child was reconstructed while a gastrostomy tube remained in place for feeding purposes and as a precaution against anastomotic complications. There were five suture lines, namely: (1) upper esophagus to cecum, (2) distal stump of ileum, (3) appendiceal stump, (4) transverse colon to stomach, and (5) ileum to transverse colon. The condition of each child was good. The chief blood loss occurred during abdominal incision and was replaced through the siphonous cannula. Operative time was approximately four hours in each case.

DISCUSSION

Primary end-to-end esophageal anastomosis continues to be the best reconstructive procedure for esophageal atresia. This report in no way indicates that we feel otherwise if the primary anastomosis can be accomplished with reasonable expectation of final success. However, if there is serious doubt regarding the viability of the anastomosis or if such is impossible, many babies can be salvaged by delay in the reconstruction procedure during saliva via cervical esophagostomy and feeding via gastrostomy tube. Delay in final reconstruction is preferable to the risk imposed by a determination to suture the two esophageal ends together when the suture line is questionable.

The complications encountered are such that one should overcome them without too much difficulty. The anastomotic leak in the neck was handled relatively easy. In this procedure there is no intrathoracic anastomosis.

which might lead to mediastinal abscess. The problem of stasis encountered in the second case was overcome clinically by reverting to frequent small feedings and gradually building up to a full three meals a day. However, cinefluorographic studies of a Lipiodol swallow showed continued slight delay of the passage of Lipiodol into the stomach and duodenum.

CONCLUSION

Two successful cases of intrathoracic transplantation of the right colon to reconstruct esophageal atresia are reported. The rationale of the procedure is discussed. The operative steps are detailed. Our initial success encourages further use of the operation in similar situations.

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HERNIATION OF THE LIVER

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HERNIATION of the liver through the right leaf of the diaphragm though still rare is being reported with increasing frequency. Although Hedblom¹ found only 14 instances of liver herniation in 857 cases of diaphragmatic hernia and only 6 additional cases had been reported up to 1951² several case reports have appeared since then.³⁻⁵ The combination of increasingly severe trauma associated with automobile accidents and the more expeditious and adequate treatment of the attendant shock, with survival of the patient makes it likely that this condition will be increasingly encountered. Likewise chest surveys of the population and routine hospital admission films will undoubtedly bring to light many previously undiagnosed and as yet asymptomatic cases. The position and appearance of the herniated liver are such as to suggest an intrathoracic tumor either of the lung or diaphragm and the diagnosis itself though suspected may be difficult to confirm without thoracotomy. In the past six years we have had the opportunity of treating four proved cases of hepatic herniation. Each of these posed a different problem and from this experience we feel certain conclusions can be drawn.

CASE REPORTS

CASE 1—H. I. W., a 55-year-old white man was admitted to the Oakland Veterans Administration Hospital on June 30, 1947. While crossing a highway on June 14, 1947 he was struck by an automobile and was hospitalized at a county hospital where a compound fracture of the right tibia and fibula was treated. He was unconscious and when his condition permitted he was transferred to this hospital. On the Orthopedic Service x-rays revealed fracture of the right tibia and fibula in poor position and these were corrected. A routine chest film incidentally revealed elevation of the right leaf of the diaphragm and an oval density just below the level of the ninth rib in the cardiophrenic angle. Because of the patient's orthopedic condition no further attention was paid to this finding until November, 1947 when he began to complain of sharp pain in the right upper quadrant which was severe and colicky. He had no pain in the left thorax. This suggested the possibility of a cholecystitis and

Fig 1



Fig 2

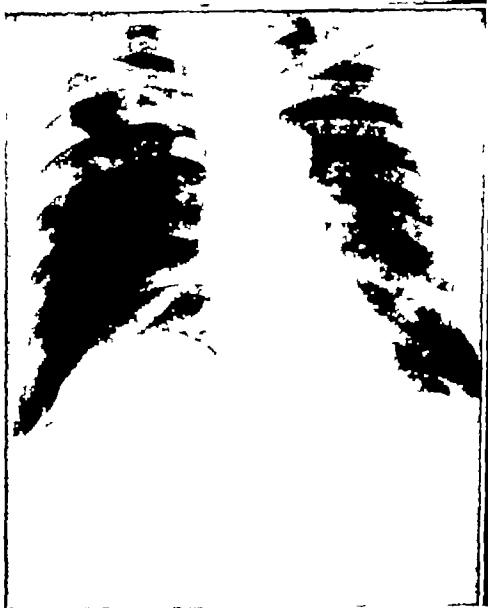


Fig 3



Fig 4

Figs 1 and 2—Case 1 Admission x rays Note presence of mass in right chest

Figs 3 and 4—Case 1 Following diagnostic pneumoperitoneum Note right pneumothorax in Fig 3 and characteristic mushroomlike mass in Fig 4

of the liver. There was a little difficulty encountered in clearing up the neck of the diaphragmatic opening, which measured about 4 inches in length and ran in a diagonal direction from the cardiac angle laterally and anteriorly. Some difficulty was experienced in reducing the herniated portion of the liver. Superior to the constriction, the liver had a grayish appearance caused by interference with the circulation. The herniation was reduced and the opening of the diaphragm closed with mattress sutures. Prior to this procedure the phrenic nerve was crushed over the pericardium for purposes of obtaining better relaxation. An intercostal catheter was placed in this space for drainage purposes. The patient made an uneventful recovery and when last seen at this hospital, in 1948, had no complaints referable to his chest or abdomen.

CASE 2—F. C., a 39-year-old white man, was admitted to the Oakland Veterans Administration Hospital on June 4, 1951, complaining of left costovertebral pain associated with fever. He had a history of renal calculi dating back to 1937 and had had several previous hospitalizations for the same complaint. His past history revealed that in 1944 he was thrown from an automobile and sustained a head injury and fractures of both clavicles, several ribs, and the right pubic ramus. In 1945 he was hospitalized elsewhere for bilateral nephrolithiasis and a right nephrolithotomy was done. During this procedure the right pleura was accidentally nicked and immediately repaired but a pneumothorax resulted and a small mass adjacent to the right heart border which had been overlooked on the routine film became apparent. A barium enema was done which showed no connection between the chest lesion and the colon. No further studies were made at that time. He reentered that hospital on Sept. 19, 1949, for further investigation of his chest lesion. A gastrointestinal series and chest fluoroscopy revealed a sharply circumscribed rounded mass overlying the right diaphragm medially and anteriorly, apparently attached to the diaphragm, but not to the heart. It was of uniform density throughout and formed no part of the gastrointestinal tract. He was discharged as "tumor of the diaphragm, nature undetermined." At the time of admission to this hospital the chest x-ray (Figs. 5 and 6) showed that the mass had increased in size though it still was asymptomatic. Bronchoscopy was entirely normal, and a diagnostic pneumoperitoneum showed air under the right diaphragm (Figs. 7 and 8) but not in the chest or above the mass. On July 10, 1951, a thoracotomy was performed. Under endotracheal anesthesia with the patient supine and the right side slightly elevated an incision was made through the fifth intercostal space. Rib spreaders were inserted. The mass seen by x-ray proved to be a central diaphragmatic hernia 5 cm. in diameter through which incarcerated liver projected. The neck of the hernia was very tight and adherent. There was no defect through which air could pass, thus accounting for the inability to demonstrate the hernia on pneumoperitoneum. The liver was freed from adherent diaphragm and during this procedure a small rent was made in the liver capsule. This was filled with Gelfoam and sutured in place by Atraumatic cut-out. The liver was returned to the abdomen and the diaphragm repaired by overlapping the edges with mattress suture of No. 20 cotton. The phrenic nerve was crushed and intercostal drainage tube placed dependently and connected to an underwater seal and the incision closed in layers with cotton. The patient made an uneventful recovery.

Fig 5

Fig 6

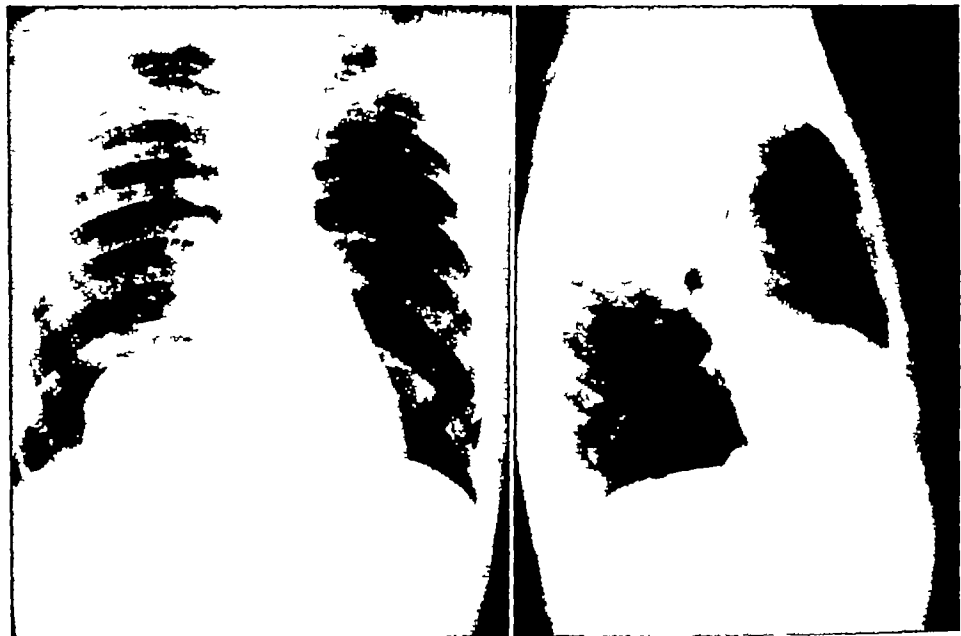


Fig 7

Fig 8

Figs 5 and 6—Case 2
Figs 7 and 8—Case 2
mass on lateral view Fig 8

Note large mass in right chest adjacent to the heart.
After pneumoperitoneum has been induced Note mushroomlike

no bright blood nor pus in the stools. In 1943 an episode of dysentery occurred for which he was hospitalized at the San Diego Naval Hospital for a period of one month. From 1943 on, the patient continued to have three to four soft, nonbloody stools daily, but every two months or so there would be a recurrence of diarrhea with passage of from six to eight watery loose stools daily. Each bout would last anywhere from one to three days, subsiding spontaneously. The episodes seemed to be exacerbated by overindulgence in whiskey or the ingestion of improper food. At the age of 2 years, the patient had been run over by an automobile while lying face down on the ground. The wheel was stated to have passed over the lower portion of his chest. Although he recalled no definite details of this event, he was told by his mother that he was very sick at the time and had been hospitalized for ten days.



FIG. 9



FIG. 10

FIG. 9 and 10—Chest. Note mass in postero-lateral aspect of right chest.

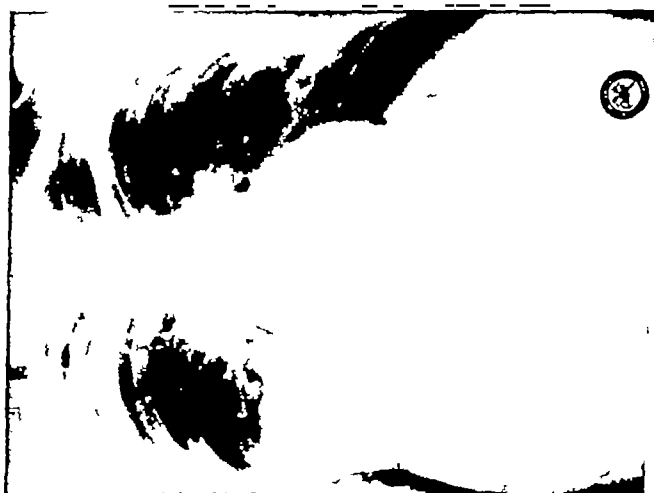


Fig 11



Fig 12

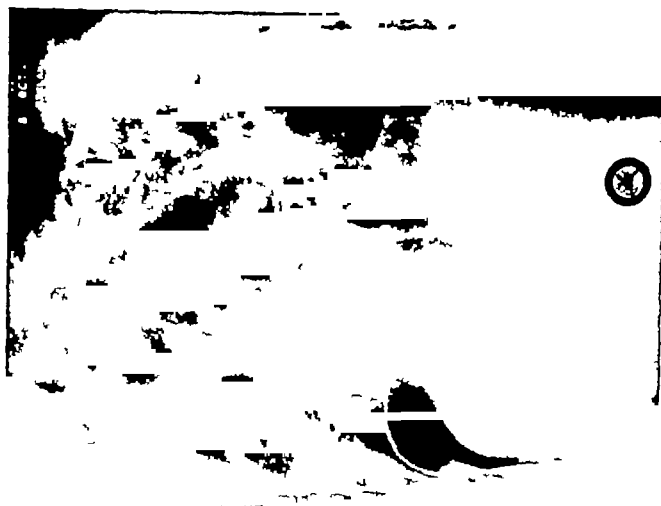


Fig 13

Figs 11 12 and 13—Case 3 The pneumoperitoneum emphasizes the mushroomlike mass representing the herniated liver

and the right coronary ligament, which was adherent to it. The sac of the hernia was trimmed at its adherent portion about the neck and the dome of the liver. The liver was returned to the abdominal cavity and the diaphragm closed over by mattress sutures. Palpation of the liver revealed no abnormalities. His postoperative course was uneventful and he was discharged from the hospital on Nov. 30, 1953.

CASE 4—G. A. C., a 59-year-old white man, was admitted to the Oakland Veterans Administration Hospital on Feb. 23, 1954. He complained of weakness, anorexia, weight loss, and for the past six months, an irritating hacking nonproductive cough with exertional dyspnea. In 1945 he was in an automobile accident sustaining fractures of the eighth and ninth ribs on the right, and requiring a thoracotomy. He had previously been hospitalized elsewhere from Feb. 27, 1949, to April 18, 1949, at which time an x-ray of the chest revealed old fractures of the right eighth and ninth ribs posteriorly, and a mass at the base of the right lung. No diagnosis of the mass was made. At the time of admission to the Oakland VA Hospital an x-ray of the chest (Figs. 14 and 15) disclosed the presence of a mass on the



Fig. 14



Fig. 15

Figs. 14 and 15—Case 4. Note mass in right chest.



Fig 16



Fig 17



Fig 18

Figs 16, 17 and 18.—Case 4. Pneumoperitoneum reveals the mushroomlike mass enclosed in a hernial sac. This is best demonstrated in the internal view (Fig 17) and the oblique view (Fig 18).

DISCUSSION

Etiology—The etiological factor in all our cases was severe trauma to the thorax usually associated with multiple rib fractures. Although the history of trauma may be readily obtained even after many years, in one instance (Case 3) only after close questioning was this information elicited. Congenital defects in the right leaf of the diaphragm may allow the liver to herniate through it and such cases have been reported.⁶ The probabilities are that such herniations would produce symptoms and be discovered long before the individual reached adult life.

Symptoms—It is interesting to note how long the patient can remain symptom free after the trauma occurs. The time interval between the traumatic episode and surgery varied in our cases from five months to thirty-three years. In the case reported by Child and his associates,² the traumatic hernia had occurred forty-four years prior to operation although gastrointestinal symptoms had been present for only twelve years. Similarly in the case described by Knopp³ trauma had occurred eighteen years before surgery. Symptoms occur only when the mass increases sufficiently in size to produce cardiopulmonary embarrassment³ or if sufficient constriction occurs then right upper quadrant pain or substernal distress may result due to interference with the blood supply and the production of early strangulation of the liver substance. This has been reported by Wolfson and Goldman.⁴ Similarly in our first case definite impairment of circulation was noted at the time of operation. The liver had a grayish color which rapidly disappeared when the constriction was freed. A biopsy of the liver at the time of surgery revealed normal liver parenchyma. It may be that the onset of symptoms long after herniation has occurred is caused in part by the descent and flattening of the diaphragm which occurs with aging and accompanying emphysema.

Diagnosis—A careful history inquiring as to the possibility of severe trauma is most important. Frequently the injury has taken place so many years ago that the patient fails to recall it or feels it has no connection with his present complaints. Such was the situation in Case 3 where after the diagnosis was entertained careful questioning of the patient revealed a history of severe trauma at the age of 2 years. The presence of a mass as seen by x-ray for a period of years which has been relatively asymptomatic is not entirely self-evident and should also make one suspicious of this possibility. In every instance a characteristic picture of a circumscribed rounded mushroomlike mass lying above the diaphragm and intimately associated with the right heart border is seen on x-ray. It has been suggested by numerous authors⁷⁻⁹ that pneumoperitoneum is an excellent tool in the diagnosis of this condition. Certainly if pneumoperitoneum is induced and air enters the thorax a rent must be present in the diaphragm. This is well demonstrated in Case 1. It must be emphasized, however, that if the condition is of long standing and a tight constriction is present around the herniated liver no air can pass through into the chest. Fibrous adhesions between the diaphragm and visceral pleura may also prevent the accumulation by forming a seal (Case 4). It is therefore

stressed that while this test may be of positive value in demonstrating a communication between the abdomen and thorax, the absence of such communication does not in any way exclude the diagnosis. Our second, third, and fourth cases demonstrate this point very well. Pneumoperitoneum, however, does emphasize the characteristic mushroomlike mass which is seen on the x-ray.

Treatment—The treatment of this condition is the same as that for any other hernia. A thoracotomy should be performed, the liver returned to the abdominal cavity, and the defect repaired. This is a simple procedure, does not require prolonged hospitalization, and definitely establishes the diagnosis. The symptomatic case requires immediate surgery because of the possibility of strangulation. This is well-demonstrated by Case 1 where definite impairment of blood supply to the herniated liver was noted at the time of operation with immediate return of normal color when the herniation was reduced. In our second case, the lesion had been diagnosed elsewhere as "tumor of the diaphragm" and caused the patient considerable anxiety. The impossibility of making an absolute diagnosis without thoracotomy led to the repair of the hernia and the relief of the patient. In our third case the patient had been treated on numerous occasions for amoebic abscess because of a history of dysentery while serving in China in 1937 and the presence of a "bump" on his liver as seen by x-ray. In fact, his complaint of "something pushing up on the right side of the chest" was interpreted as increasing tension of an hepatic abscess. The failure by numerous physicians to properly establish the correct diagnosis led to frequent periods of hospitalization, loss of work, and recurrent courses of emetine. The fourth case had frequent admissions to hospitals with vague symptoms referred to the right upper quadrant of the abdomen, and without a proper diagnosis.

We do not feel that this lesion should be allowed to remain because it is asymptomatic at the time of discovery. Certainly our experience as well as that of others has shown that symptoms do become apparent and troublesome years after the lesion is first found.

SUMMARY AND CONCLUSIONS

The presence of an asymptomatic intrathoracic mass in the right lower chest poses a problem in differential diagnosis. One of the conditions to be considered is hepatic herniation. This is rare and, in adults, usually follows severe trauma. It may be asymptomatic for years, first being discovered during the course of a routine chest x-ray. Occasionally symptoms occur consisting essentially of pain in the right upper quadrant of the abdomen or across the right lower chest. This may grow in intensity and is usually secondary to the increasing size of the mass with encroachment on neighboring structures or strangulation of the liver. In practically every instance the x-ray presents a typical appearance, that of a circumscribed, rounded, mushroomlike mass lying above the diaphragm and intimately associated with the right heart border. Though pneumoperitoneum may help in establishing the diagnosis in early cases, frequently the rent in the diaphragm is sealed off by the large mass of liver protruding through it. However, the use of pneumoperitoneum

does serve to emphasize the characteristic x-ray appearance of the lesion. Barium enema, gastrointestinal series, chest fluoroscopy and bronchoscopy are useful in excluding other diagnoses. A careful history inquiring into past severe trauma is of the utmost importance. Since these lesions tend to increase in size over the years, eventually producing symptoms and the possibility of missing a tumor is always present, it is urged that all suspected hepatic herniations be explored and corrected.

We are indebted to Miss Maude Greenwood, Chief, Medical Illustration Laboratory, and to Mr. Jack F. Shipley, medical photographer, for the x-ray reproductions.

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FIBROSARCOMA OF THE BRONCHUS

REPORT OF A CASE

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PRIMARY fibrosarcoma of the bronchus is a lesion of considerable rarity. Whereas in the older medical literature reports of pulmonary fibrosarcoma were quite frequent, it is now known that the great majority of these reports actually represented undifferentiated carcinomas, or lymphosarcomas of the mediastinum with extension to the lung.

Adler,¹ in his book published in 1912, listed ninety cases of pulmonary sarcoma. The lack of accurate pathologic description made it impossible to establish a single one of his cases as being a primary fibrosarcoma of the bronchus.

In a review of the literature in 1931, Ball² found fourteen cases of pulmonary sarcoma and added one case of his own. In a detailed analysis of these cases, however, only the cases of Ball² were found to be adequately authenticated. But even these two cases so extensively involved the lung that their exact sites of origin could not be defined accurately.

In 1950, Black³ was able to find only five authenticated cases of primary fibrosarcoma of the bronchus in the entire medical literature. To these he added one case of his own. In the same journal in which Black's paper was published, Carswell and Kraeft⁴ reported an additional case.

Stout⁵ has greatly clarified the somewhat confused picture of fibrosarcoma. He has pointed out that the tumor is primarily characterized by the formation of collagen and reticulin fibers in varying amounts. The individual cells are usually spindle shaped, and mitotic figures are rare since the tumor is usually slow growing. The presence of giant cells is uncommon.

Stout⁵ recognizes two forms of fibrosarcoma, the well-differentiated and the poorly-differentiated. In the well-differentiated tumors the collagen and reticulin fibers are wrapped around the cells, and these bundles or cords of spindle shaped cells and fibers are usually arranged in gentle curves, but abrupt changes in direction of these cords may occur giving the appearance of whorls on cut section to which some authors have given the name "neurogenic fibrosarcoma." The cells of the well-differentiated tumors are uniform without much hyperchromatism, with well-developed collagen and/or reticulin fibers and few or no mitoses. In the poorly-differentiated tumors the cells are closely packed with little or no collagen and only delicate reticulin fibers. There is some hyperchromatism, and mitoses are frequent.

Of the five cases of fibrosarcoma of the bronchus reported by other authors and listed by Black,³ in only one was pulmonary resection attempted. The pa-

tient however died during surgery of pulmonary hemorrhage. In another case, no treatment was given. In the three remaining cases the lesion was removed endoscopically. In Black's case and in the case reported by Carswell and Krieger⁴ pneumonectomies were performed.

Gelsner, DeBakey and associates⁵ reported one case of a neurofibrosarcoma removed by pneumonectomy with a survival of twelve years, but the site of origin of the tumor was not given. Stout⁶ would classify this as a fibrosarcoma. Mallory⁷ reported two cases of fibrosarcoma in which pneumonectomy was performed but in each case it could not be definitely established whether the lesion was primary or metastatic.

The paucity of reports of proved pulmonary fibrosarcoma leads us to report a case which we believe to be a well-verified primary fibrosarcoma of the bronchus for which a pneumonectomy was performed with survival of thirteen months to date.



Fig. 1. Anteroposterior (A.P.) chest plate showing a rounded shadow in the left lower lung zone.

interlobar fissure and lingula. A biopsy of the mass was taken which was reported on frozen section to be malignant. A pneumonectomy was therefore performed. The patient's postoperative course was entirely uneventful, and he was dismissed from the hospital on the tenth postoperative day.

At present, the patient is alive and completely asymptomatic, thirteen months following surgery.



Fig 2—Gross photograph of resected specimen. The lumen and the involved bronchus have been sectioned longitudinally, and the halves spread apart. The tumor surrounds and causes stenosis of the segment of bronchus seen passing horizontally across the upper third of the figure.

Gross and Microscopic—The gross specimen consisted of a lung weighing 260 grams. There was a tumor mass arising from the superior branch of the lingular bronchus. This mass was demarcated from the surrounding tissue but not encapsulated, was roughly spherical in shape. It measured 2.5 cm in diameter and completely surrounded the bronchus, which was eccentrically placed in the tumor. There was a marked stenosis of the bronchus with ulceration and loss of bronchial mucosa (Fig 2). Section of the mass showed it to be yellow white in color, moderately firm, and noncystic, with masses of tumor tissue organized in whorlike formation.

Microscopically, the tumor showed numerous bundles of elongated fusiform cells which interlaced and anastomosed freely with each other. In the individual fascicles the cells showed a mild degree of pleomorphism, although the majority of cells were spindle shaped. Many nuclei were markedly enlarged, hyperchromatic, and showed bizarre configurations. Abnormal mitoses were scattered in small numbers throughout the tumor. The bronchial mucosa was absent, the bronchial surface being made up of tumor tissue. Beneath the surface a few bronchial glands could be seen surrounded by infiltrating tumor (Figs 3 & 5).

A Masson stain demonstrated the production by the tumor cells of a small amount of fibrillar intercellular collagen, and a Wilder stain showed a large amount of reticulum throughout the entire tumor.

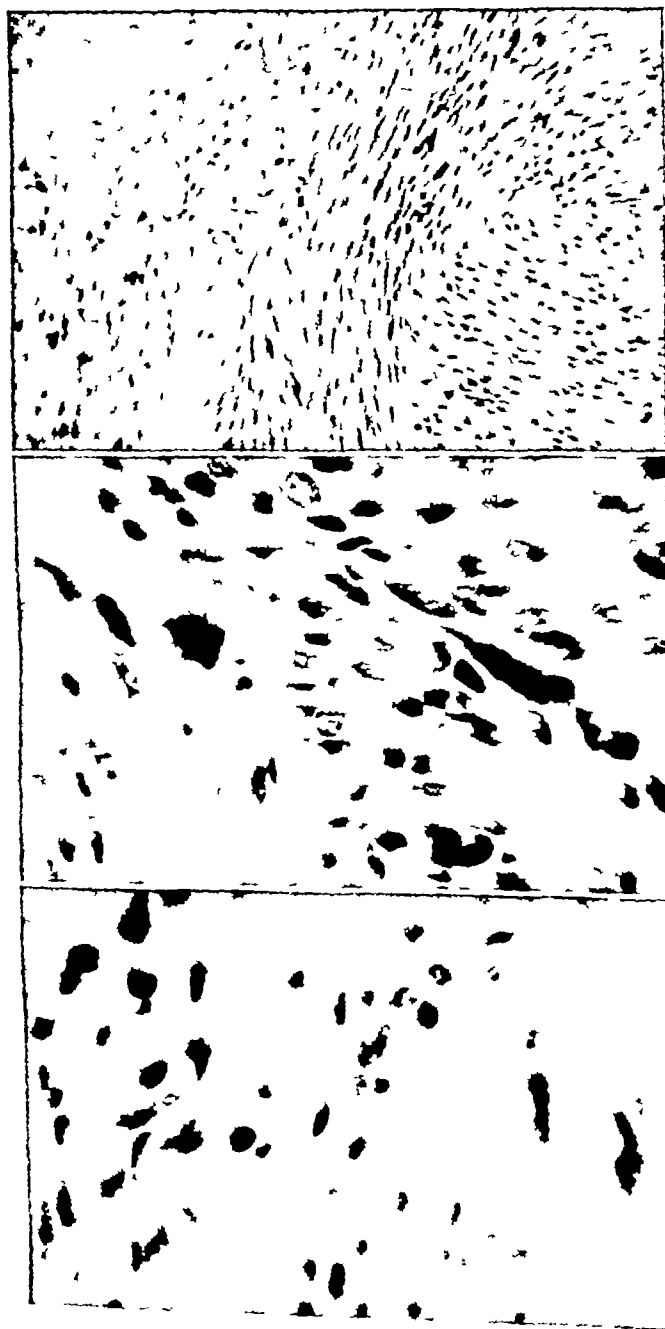


Fig 5

Fig 1

Fig 3

Fig 3.—Fibrosarcoma of the lung. Note fasciculation. Palsading of nuclei is present in the longitudinally cut fibrous tissue bundle. (Magnification $\times 100$ reduced $\frac{1}{2}$.)
 Fig 1.—Fibrosarcoma of lung, showing nuclear pleomorphism and faint hyperchromatic nuclei. (Magnification $\times 170$ reduced $\frac{1}{2}$.)
 Fig 5.—Fibrosarcoma of lung, abnormal mitoses. (Magnification $\times 170$ reduced $\frac{1}{2}$.)

DISCUSSION

We are in accord with Black³ in believing that these tumors, despite their often slow growth, should be removed by pulmonary resection. Endobronchial removal of these lesions, when feasible, in no way assures their complete removal. As in other neoplastic diseases of the lung, radical surgery provides the best hope for cure.

SUMMARY

1. A brief résumé of the medical literature on fibrosarcoma of the bronchus is presented.

2. A case of primary fibrosarcoma of the bronchus, treated by pneumonectomy, is reported. The patient is alive and well thirteen months postoperatively.

3. It is believed that pulmonary resection is the rational method of treatment of pulmonary fibrosarcoma.

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THE CAUSES OF DEATH FOLLOWING ESOPHAGEAL RESECTION FOR CARCINOMA

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THE surgical treatment of primary epidermoid carcinoma of the esophagus has been found to be extremely unsatisfactory in our experience. With few exceptions all of our patients with carcinoma of the esophagus have presented themselves in a moderately severe to severe state of dehydration and malnutrition. A high proportion of the tumors are situated in close proximity to the region of the aortic arch and the bifurcation of the trachea. The technicalities involved in the extirpation of tumors in this region when combined with restoration of the alimentary tract in a single stage operation are more complicated than in the less frequently involved terminal esophagus. Even so, the salvage rate might be more presentable were it not for the fact that the operative mortality rate has been very high. In those surviving, the long-term results have been very poor.¹⁰ Others^{11, 12, 13} have reported a somewhat similar experience.

In an attempt to find some reason or reasons for the extremely poor results, we have analyzed our cases with particular regard to the causes of death following treatment by resection.

In the 13½ year period between Jan. 1, 1940, and June 30, 1953, 210 patients were observed with histologically proved epidermoid carcinoma of the esophagus. We have observed no cases of adenocarcinoma primary in the esophagus. No case of adenocarcinoma of the stomach invading the esophagus is included in this series.

The relative incidence of primary epidermoid carcinoma of the esophagus and primary malignancies in the stomach, colon, rectum and other sites has been reported in an analysis of an earlier series.¹⁰ In our experience carcinoma of the esophagus is the most common malignant neoplasm in the alimentary tract. Also in the same report observations were made on other features of carcinoma of the esophagus, notably its great preponderance in the Negro, the high incidence of involvement of the region of the arch of the aorta and the methods of diagnosis, treatment and results.

The uppermost levels of involvement of the esophagus in 199 cases in which it could be determined accurately are shown in Table I.

TABLE I. LEVEL OF INVOLVEMENT

TABLE II OPERABILITY AND RESECTABILITY RATES

Cases	210
Inoperable	138 (66%)
Operable	72 (34%)
Nonresectable	29 (40%)
Resectable	43 (60%)
Palliative	32 (74%)
Curative	11 (26%)

The operability and resectability rates for the 210 cases are shown in Table II. The reasons for inoperability were the wide extent of the local lesion (such as invasion of the trachea or of a bronchus), distant metastasis, or both, or severe malnutrition without improvement on therapy. Of the resectable cases, only one in four could be considered to have had a theoretically curative resection. The 11 cases having a theoretically curative resection represented only 5.2 per cent of the entire group of 210 cases.

All of the cervical lesions explored were found to be nonresectable.

There were 43 cases with involvement of the thoracic esophagus treated by resection. Early in the series there were 4 cases treated by the Toirek procedure. The remaining 39 cases were treated as described here. In one case, a two stage operation was performed. At the first stage, the stomach and esophagus were mobilized through a median sternotomy in continuity with a vertical upper abdominal incision. At the second stage, using a right thoracotomy approach, an esophagogastrostomy was performed above the level of the arch of the aorta. In one case, a right anterior thoracotomy approach was used, after mobilization of the stomach through a separate abdominal incision, with restoration of continuity by an intracervical esophago-esophageal anastomosis. The remaining 37 patients were treated by restoration of the continuity of the alimentary tract in a single-stage operation through a left thoracotomy approach, using the stomach to replace the resected portion of the esophagus, except in three cases, as follows: an esophagojejunostomy in the neck was performed in one, a plastic tube¹ was used to replace the resected portion of the esophagus in another, and an esophago-esophagostomy in the thorax without transplantation of the stomach² was performed in another. None of the patients treated by resection had had a preliminary gastrostomy.

The mortality rates in the cases treated by resection are shown in Table III.

Before proceeding with an analysis of the causes of death following resection, it must be said also that in the 29 cases which were operable but non-resected, there were 5 deaths as a direct result of the operation and 2 deaths after recovery from the operation but prior to discharge from the hospital. The mortality rate as a direct result of the operation was 17 per cent. The causes of death in the 5 cases were unrelieved tracheal obstruction in one, postoperative shock and empyema in one, uncontrolled hemorrhage due to laceration of the aorta in one, blood transfusion reaction in one, and hemorrhage from the trachea in the presence of a tracheotomy in one. The causes of death in the 2 cases in which death occurred following operation and before discharge from the hospital was inanition.

TABLE III. TYPES OF RESECTION AND ANASTOMOSIS FOR THORACIC LESIONS

	CASES	SURVIVED	DIED	MORTALITY RATE (%)
Total	47	19	28	56 average
I E = Esophago-esophageal	4	1	3	75
I E = Esophago-gastrostomy	1	0	1	100
I G = Esophago-gastrostomy	2	2	4	50
I A = Esophago-anastomosis	1	0	1	100
I G = Esophago-gastrostomy	11	7	6	55
I G = Esophago-gastrostomy	6	2	4	66
I G = Esophago-gastrostomy	10	6	4	40
Pleurostomy	1	0	1	100
I E = Esophago-esophageal	1	1	0	0

I E = Esophago-esophageal

I G = Esophago-gastrostomy

I A = Esophago-anastomosis

The causes of death in the 24 cases of carcinoma of the thoracic esophagus treated by resection are shown in Table IV. It will be noted that in 11 cases empyema developed on the operative side. Most often this was due to failure of healing of the anastomosis with esophagopleural fistula. The resulting empyema did not improve following the institution of adequate drainage and discontinuance of oral feedings. Parenteral feedings or jejunal feedings were substituted. The animal feedings were given either by a tube passed transnasally into the jejunum or by jejunostomy.

The high mortality rate following resection warrants a review of the status of the patients on admission and of our methods of preoperative therapy, operative technique, and postoperative therapy.

TABLE IV. CAUSES OF DEATH FOLLOWING RESECTION OF THE THORACIC ESOPHAGUS FOR CARCINOMA

	CASES
At pneumonia	9
Empyema (pleurostomy and empyema)	1
Empyema with anastomosis intact	1
Esophagopleural fistula	2
Esophagopleural fistula and chronic pyelonephritis	2
Chronic pyelonephritis	1
Acute pyelonephritis	1
Acute renal failure	1
Acute renal failure	1
Acute renal failure	1
Total	24

weight loss expressed in terms of the percentage of total body weight averaged 17 per cent, with extremes of zero and 43 per cent. In contrast, in the 19 patients who survived resection, there was no significant difference in the same observations.

Many cases showed hemoconcentration before hydration, as one would expect. After hydration and before blood transfusions, anemia was present in 14 of 24 patients who died following operation. Anemia was present in the same degree in those who survived resection.

Abnormalities in the serum protein determinations were noted in 8 of the 22 patients in whom determinations were made, among the 24 patients who died following resection. There were noted a reduction in the total serum proteins in 4 cases and a reduction in the albumin component in 7 cases, varying from slight to marked, with reversal of the albumin-globulin ratio in some. In contrast, among the 19 patients who survived resection, serum protein determinations were available in 12. In none of the 12 cases was there a reduction in the total serum proteins, but in 7 of the 12 cases the albumin was reduced, with reversal of the albumin-globulin ratio in several.

Serum sodium, potassium, and chloride determinations were normal in all patients on whom such determinations were made, except in one in whom hyponatremia and hypokalemia were present on admission. This condition was corrected easily and promptly.

In addition to carcinoma of the esophagus, a total of 17 diseases was found in 16 of the 24 patients who died following operation. Hypertension and significant arteriosclerosis were found in 4 cases, uncomplicated hypertension alone in 2 cases, diabetes in 1 case, unexplained hepatomegaly in 1 case, nephrosclerosis with a blood urea nitrogen of 30 mg per cent in 1 case, transient pneumonia in 1 case, chronic osteomyelitis of the femur in one case and chronic alcoholism in 1 case. None of these was considered to be a contraindication to operation, and in none who died was there any concrete evidence that any of these abnormalities had contributed to death.

In contrast, in the 19 patients who survived esophageal resection, there were 6 patients with diseases other than carcinoma of the esophagus. Two had hypertensive cardiovascular disease, one had pulmonary emphysema not clinically significant, one had a positive Wassermann, one had granuloma inguinale, and one had a nodular goiter which was removed prior to esophageal resection.

In only 1 of the 24 patients who failed to survive resection was there any evidence prior to operation of extension of the tumor beyond the esophagus which could be ascertained by clinical or accessory examination. The one case had a firm palpable supraclavicular lymph node which was not biopsied.

PREOPERATIVE THERAPY

Restorative therapy consisted of the administration of intravenous fluids, or of forced feedings via a transnasal intragastric Levin tube supplying proteins, fats, carbohydrates, and vitamins with provision of as many calories as possible, of blood to correct anemia, and of treatment of associated diseases when indicated. Therapy was continued from 4 to 61 days preoperatively in

the 24 patients who died following resection. The average duration of therapy in those who died was 17 days. The average duration in those who survived was 16 days, with extremes varying from 2 to 30 days.

Despite this period of preoperative therapy, the mean weight gain was only 1 pound in the 24 patients who died following resection. The extremes were loss of 5 pounds and gain of 10 pounds. The 19 patients who survived following resection also showed no weight gain in the average case, with variations from 10 pounds lost to 8 pounds gained. However, both groups showed a significant preoperative gain in strength and general appearance by clinical appraisal which could not be related to the weight changes.

Hormonal therapy preoperatively was used in only 1 of the 43 cases treated by resection. Testosterone was used in this one in an effort to produce a positive nitrogen balance, but this patient died. ACTH and cortisone were not used.

OPERATIVE TECHNIQUES

The operative technique for the Torek procedure need not be described. With the exception of the first case operated on after World War II (1946), the Torek procedures were performed during the war in the early periods of assessing the surgical treatment of carcinoma of the esophagus in this institution.

The operative technique used in the majority of cases has been described in a previous report¹ and the precise operations performed in all cases in this series have been enumerated. The method of anastomosis used was based on earlier experimental work.²²

In most cases, blood was given intravenously throughout operation. In those patients who died following resection, the amount transfused averaged 2,000 cc. with extremes of zero in 2 cases and 4,500 cc. in 2 cases. In comparison, in those who survived resection, the average amount of blood transfused during operation was 1,800 cc.

Intravenous fluids other than blood administered during operation were recorded in 22 of the 24 cases where death occurred following operation. The amounts varied from zero in 4 cases to 2,000 cc. in 1 case, the average being 600 cc., usually glucose in distilled water.

The exact duration of the operation among those who died was recorded in 19 cases and varied from three to nine hours, the average being five and one-half hours. The average duration of operation among those who survived was precisely the same.

Complications during operation occurred in 3 of the 24 patients who died following operation. There were severe blood transfusion reactions. In one the patient, a young type AB Rh positive, was given a total of 2,000 cc. of blood transfused. The patient was AB Rh positive, Group O Rh positive, and Group O Rh negative. A fatal reaction developed with bleeding from all wound surfaces, and death occurred in the operating room. In another incompatible blood was given to a patient who was AB Rh positive. In a third case, death occurred during operation. The patient was AB Rh positive and died four hours as a result of a course of chemotherapy. The patient was AB Rh positive and died four hours as a result of a course of chemotherapy.

POSTOPERATIVE THERAPY

Postoperative therapy consisted of intravenous alimentation, antibiotics, and constant gastric drainage by suction via a transnasal Levin tube. In general, a total of 3,000 c.c. of fluids was given daily, containing glucose, amino acids and vitamins. In retrospect, this was excessive in some patients, although it was difficult to ascertain the exact number. The antibiotics used most commonly were penicillin and streptomycin. In the few patients operated on prior to their introduction, the sulfonamides were used. The Levin tube was removed generally two or three days following operation.

Esophagograms using Lipiodol were made generally four to six days following operation. If there was no extravasation of oil, oral intake was allowed immediately in increasing amounts with a corresponding decline and abandonment of the intravenous alimentation.

Other than in the 5 patients who died within eight hours following resection, the average duration of life following resection in the remaining 19 cases was 11 days, with extremes of 2 days and 31 days.

LONG-TERM RESULTS

In addition to presenting the dire results of the operative treatment of the cases of primary carcinoma of the esophagus in this series, a word should be said about the equally dire results in the entire group. Of the 210 cases, not including one case in which treatment had not been begun at the time of this compilation, there are only 10 patients living. Three of the 10 patients were treated by resection, having esophagogastrostomy performed in the neck, at the level of the arch of the aorta, and below the arch of the aorta in one case each. The patient with the intracervical anastomosis has now survived two years and seven months, with ability to eat and swallow normally throughout but with known residual carcinoma in the lower trachea and both main bronchi. She has been treated recently by radiation therapy with a dramatic improvement in her dyspnea to such an extent that she has been able to resume her usual occupation as a domestic servant. The patient with the juxta-aortic anastomosis has now survived five years with no evidence of persistent or recurrent carcinoma to date.

Seven of the 10 surviving patients have had radiation therapy, the longest surviving patient having completed treatment only eleven months ago. However, only 3 of the 7 have a chance of a good result. The remaining 4 patients have uncontrolled primary lesions, and each has a gastrostomy for feeding purposes.

DISCUSSION

As the problem of the surgical treatment of epidermoid carcinoma of the esophagus stands at present, the operative mortality must be reduced to acceptable levels. Otherwise, serious consideration must be given to the abandonment of the procedure of esophageal resection with restoration of the continuity of the alimentary tract in a single stage operation. The possible and probable exception to this statement is in the treatment of epidermoid carcinoma in the terminal fourth of the thoracic esophagus, in which cases we believe that opera-

tion is still the preferable method of treatment. The statement applies in particular to the treatment of lesions which require superior mediastinal or intra-cervical anastomosis after their resection. The results reported with radiation therapy¹ and plastic tube replacement² serve to augment this conclusion.

An obvious need for the reduction of operative mortality is a method for the conversion of these extremely malnourished patients from a catabolic phase into a prolonged anabolic phase. At times the catabolic phase is striking even in the absence of a high degree of esophageal obstruction. The provision of calories which have been given to the present group of patients in quantities of 3 500 to 4 500 per day in the preoperative period has not resulted in the desired anabolism. What other factors are concerned in the continuation of catabolic processes in the presence of adequate caloric intake should be investigated. The duration of the preoperative preparation may be a factor of considerable importance. Prolonging this period may result in the patient's conversion to an anabolic phase but this is doubted. The possible continued growth of the neoplasm during the preoperative period serves to limit its duration unless therapeutic measures were being directed toward the neoplasm itself. Perhaps this could be achieved by radiation therapy during the interval of preparation for operation combining the two methods of treatment. The use of hormonal therapy³ in patients who fail to revert from catabolic metabolism may help to lessen the operative mortality.

Another need is revision of the operative procedure as we have performed it in the majority of cases. The approach through the left chest with trans diaphragmatic mobilization of the stomach is too time-consuming. The utilization of plastic tube replacement or of simultaneous midline abdominal and right thoracic incisions by two teams may be the answer to this portion of the problem. The present method of anastomosis of the esophagus and the stomach needs to be reviewed. The largest single cause of death in this group of patients was disruption of the suture line at the anastomosis with resultant esophago-plural fistula and empyema. Whether this disruption results from local technical causes or general causes associated with the depletion of the patient by starvation has not been determined. Both of these causes may be implicated in each case but other alimentary canal resections and anastomoses in depleted patients are not associated with such forbidding mortality. Perhaps tunneling the esophagus into the stomach by suturing the fundus of the stomach around the terminal end of the remaining esophagus would be of aid in securing firmer union between the two organs. It might be of value to mobilize all or a portion of the omentum from the transverse colon so that the omentum may be used to cover the anastomotic line. These procedures will require further investigation.

At present it is a change in the criteria for operability and resectability. Most studies have been paid to the presence of associated diseases, hypoproteinemia, and lack of weight gain during intensive restorative therapy despite the presence of a strong and good general appearance. Actually we have seen a number of patients who had succumbed to the use of x-ray therapy and who had been considered operable but who had been considered formerly

It is believed that overttransfusion of blood and overhydration during the operative and immediate postoperative periods have contributed to the fatal outcome in more cases than in which they are reported as being the primary cause of death. Replacement of blood should not exceed the loss in order that the number of transfusions may be kept at a minimum, particularly from the standpoint of reduction of the incidence of transfusion reactions, the chances of which increase geometrically with each of multiple transfusions.

SUMMARY

1 An analysis of 210 patients with primary epidermoid carcinoma of the esophagus is presented, with particular regard to a critical review of the causes of death in patients treated by resection.

2 The mortality rates for resections of the thoracic esophagus, with and without restoration of continuity of the alimentary tract, are presented.

3 There were 43 cases treated by resection of the esophagus, with an average operative mortality of 56 per cent.

4 The causes of death are enumerated, the most frequent being failure of healing of the anastomosis with esophagopleural fistula and empyema.

5 The causes of death have been analyzed from the standpoints of the status of the patients on admission, the preoperative therapy and its duration, the operative technique, and postoperative therapy.

6 The operative mortality must be reduced in order to justify the continued use of resection by the methods described, especially in the treatment of lesions in the region of the arch of the aorta.

7 Considerations on the needs for the reduction in operative mortality are presented.

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THE SURGICAL TREATMENT OF CHRONIC PERICARDIAL EFFUSION AND CARDIAC TAMPONADE

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THE operative treatment of patients with inflammatory lesions of the pericardium has been generally limited in application, during recent years, to those patients who were suffering from a constrictive type of pericarditis. Relatively few references appear in the current literature by authors who advocate pericardiectomy during the effusive stage of pericarditis, particularly of the nonsuppurative type. It may be of interest to recall that as early as 1819 Romero¹ had operated on three patients with effusive pericarditis, two of whom survived. The operation consisted of an incision made through the left fourth interspace near the sternum, the distended pericardium was felt with the finger, picked up with forceps, and cut with scissors. A number of other successful cases was subsequently reported, but due to the influence of Dieulafoy,² in 1873, pericardiocentesis replaced incision and drainage as the method of choice in the treatment of pericardial effusion. This influence still prevails. Today the treatment of pericarditis with effusion is usually carried out by the internist, who commonly employs repeated pericardiocenteses to decompress the pericardial cavity. While this regimen is usually satisfactory, we believe that it may have the following disadvantages: (1) Pericardiocentesis has, on occasion, resulted in fatal hemorrhage. (2) Cardiac tamponade usually is relieved only temporarily, and prolonged tamponade may result in serious myocardial, hepatic and pulmonary damage. (3) The longer the infection in the pericardial cavity persists the greater is the danger of progression to the constrictive phase of pericarditis. In the series of Heuer and Stewart, 27 per cent of patients with constrictive pericarditis had previous pericardial effusion.

Because of these disadvantages and because pericardiectomy is much more easily performed during the effusive rather than the constrictive stage of pericarditis, we believe that the recent suggestion by Holman³ of early pericardiectomy has great merit and that the operation is indicated, not only for patients with acute tuberculous pericarditis as recommended by him but also for some patients with idiopathic pericarditis. An important contribution which has influenced us in coming to this decision is that of Barker and Johnson,⁴ who recently emphasized the fact that nontuberculous pericarditis with effusion is a chronic disease more frequently than is generally appreciated. One of their patients suffered from a pericardial effusion for six years before the lesion progressed to the constrictive stage.

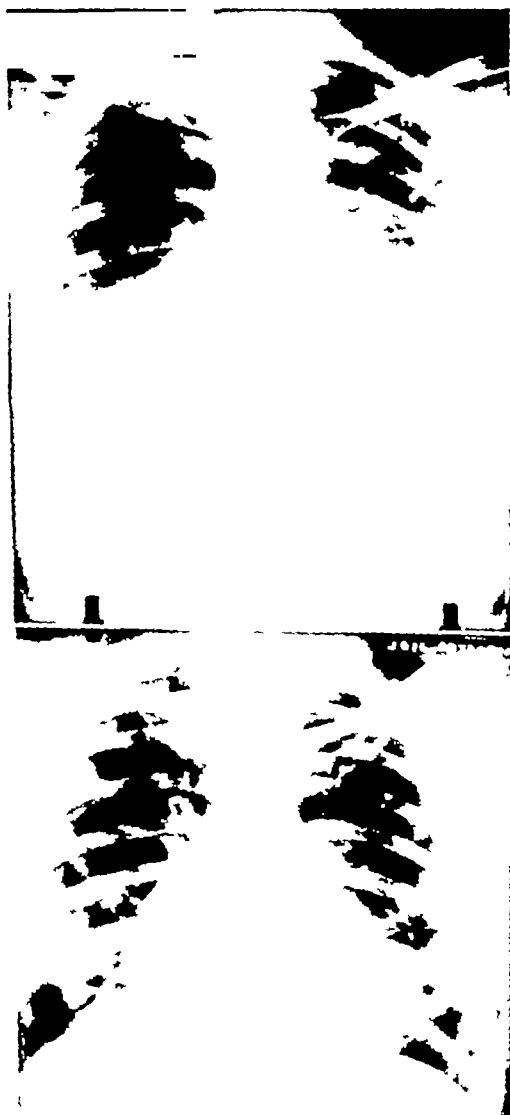
We have had an opportunity to observe the results of elective pericardiectomy performed during the stage of effusive pericarditis in four patients and also to follow the courses of two other patients from the stage of effusion to eventual constrictive pericarditis, which required a technically more difficult operation. In the latter patients, symptoms and signs of cardiac tamponade persisted unnecessarily for several years while the stage of constriction developed. We believe that both patients would have profited from an earlier pericardiectomy. One patient had tuberculous pericarditis, but in five patients the etiology was undetermined. The ages of the patients ranged from 34 to 67 years. The predominating symptom in all patients was severe dyspnea. Some or all of the following signs of cardiac tamponade were present in each case before operation: hepatomegaly, ascites, low pulse pressure, low blood pressures, elevated venous pressure, prolonged circulation time, dependent edema, and low voltage electrocardiographic patterns. The apparent duration of the disease prior to operation averaged seventeen months. Five of the six patients had moderate fevers and received streptomycin and PAS therapy for at least two weeks prior to operation.

CASE SUMMARIES

CASE 1—W. L., KCH No. 2151451, a 41 year old tailor, was admitted to the King County Hospital for the first time on May 3, 1951, complaining of cough and night sweats of three weeks' duration. The temperature was 102.5° F, the blood pressure was 140/80, the pulse was 80, and the respiration was 24. Physical examination revealed moist rales in the left lung. A roentgenogram of the chest revealed a water bottle configuration suggesting pericardial effusion (Fig. 1). A kymogram showed a decrease in amplitude of cardiac pulsations. The blood Mrazini, heterophile agglutination test, cold agglutinins, cephalin flocculation, and cholesterol determinations were all within normal limits. Two smears of the sputum for acid fast bacilli were negative. The patient was discharged on June 27, 1951, with a diagnosis of bronchial pneumonia and pericarditis with effusion. He was admitted for the second time on Sept. 28, 1951. He complained of a cough productive of a moderate amount of whitish sputum and dyspnea which had been present since his previous discharge. He had been vomiting for one week. Physical examination revealed a large area of cardiac dullness extending to the left midaxillary line, sacral and ankle edema, and hepatomegaly. The blood count was normal except for an 8 per cent basophilia. The venous pressure was 180 mm of water and the circulation time, with Decholin, was 17 seconds. Kymography revealed no visible excursion of the left side of the heart. On September 29 a pericardicentesis was performed with the removal of 300 cc of straw colored fluid. Culture of the fluid for acid fast organisms was negative. Urine cultures also were negative. Blood chemistries, including urea, sugar, cephalin flocculation, phosphorus, and alkaline phosphatase, were within normal limits. The albumin was 3.6 Gm, and the globulin was 2.5 Gm. Low voltage QRS complexes and flattened T waves were found in the electrocardiogram. The tuberculin 1:1,000 was positive. A ballistocardiogram was negative. On October 5 and October 12, 450 cc and 350 cc, respectively, of serosanguineous fluid was removed. The patient failed to improve, although he received dihydrostreptomycin for one month. On Nov. 24, 1951, the left chest was entered anterolaterally at the level of the fourth left rib and cartilage. The pericardium was 3 mm in thickness and cartilaginous in consistency. The heart was not enlarged. An anterolateral segment of parietal pericardium 7 by 8 cm was resected. The pleural space was drained and the chest closed. The patient's postoperative course was satisfactory. The pathologic report indicated a chronic fibrosing pericarditis probably granulomatous. No carcinoma cells were found in the pericardial fluid. Gradual clearing of the postoperative

pleural reaction on the left side occurred. The patient was last seen in April, 1954, at which time he occasionally had mild swelling of his ankles at the end of the day, but was otherwise well and attended work regularly. The roentgenogram of the chest was normal (Fig 2)

Fig 1



CASE 2—M W, B H No 153785, a 43 year old Negro woman, was admitted to the Brooklyn Hospital on May 27, 1952. In February, 1952, the patient had developed severe dyspnea. She had previously been admitted to another hospital, where it had been necessary during the next ten weeks to perform pericardiocentesis on seven occasions. She complained of swelling of the ankles, and cough, as well as a sensation of pressure in the left chest. There was no history of rheumatic fever. Except for pneumonia at the ages of 20 and 31, there was no significant past history.

The patient's father and mother had died of heart disease at 40 and 39 years of age, respectively.

Physical Examination—The patient was a dyspneic, orthopneic Negro woman, weighing 154½ pounds and measuring 5'3" in height. She appeared chronically ill. The pulse was 80, the blood pressure 150/120, temperature, 99° F, respiration 14.

The neck veins were distended. Motion of the left hemithorax was limited, and there were dullness to percussion and diminished to absent breath sounds over the left chest. The cardiac silhouette was greatly enlarged to percussion with regular sinus rhythm and no murmurs. The heart sounds were loud and of good quality. The liver was enlarged to three fingerbreadths below the costal margin. Ascites and ankle edema were prominent.

A diagnosis of pericardial effusion was made. On May 28, 1952, a pericardial tap was performed and 550 cc of dark red fluid was removed. On May 30 the venous pressure was 178 mm of water, which was elevated to 245 mm with right upper quadrant pressure and later returned to 182 mm of water. The circulation time, using Decholin, was 11 seconds. On June 3 a second pericardial tap yielded 450 cc of bloody fluid. On June 5 the venous pressure was 94 mm of water with right upper quadrant pressure at 102 mm. The circulation time was 12 seconds. On June 6, 1952, 950 cc of grossly bloody fluid was removed from the pericardial sac. On June 7 the liver was two fingerbreadths below the right costal margin. The neck veins were still distended. On June 15 a loud pericardial friction rub could be heard over the pericardium. The edge of the liver was palpated 4 cm below the costal margin. A pericardial tap was unsuccessfully attempted on June 15.

An electrocardiogram revealed QRS complexes and T waves of very low voltage in all leads. These complexes, particularly the T waves, became larger following removal of pericardial fluid. Five examinations of the pericardial fluid, including one culture, did not reveal acid fast bacilli.

The total protein was 5 Gm per 100 cc., the albumin was 2.4 and globulin 2.6. Plasma chlorides were 102 meq. The blood count was normal except for anemia, the hemoglobin being 65 per cent and the red cells 2,940,000. The urine analysis was normal. The erythrocyte sedimentation rate was 16 and 51 mm. on May 31, 1952, and 31 and 71 mm. on June 9, 1952, the readings being taken at half hour intervals. The blood sodium was 140 meq, potassium was 4.03 meq. Blood urea, creatinine, and sugar values were within normal limits.

Roentgenograms of the chest revealed a very striking enlargement of the cardiac silhouette, typical of pericardial effusion (Fig 3). Air was instilled into the pericardial sac revealing an apparently normal sized heart, although the cardiophrenic angles could not be visualized (Fig 4).

The patient received Mercuryhydrin on five occasions, was digitalized prior to operation, and received 1 Gm of streptomycin and 8 Gm of para-aminosalicylic acid daily, starting on June 2 and continuing until July 7.

On June 19, 1952, an extensive parietal pericardiectomy was performed. The pericardium measured approximately 3 to 4 mm of thickness. No evidence of constriction of the heart was found, but approximately 500 cc of fluid containing some fibrinous clots was aspirated from the pericardial sac. Cytologic examination of the pericardial fluid showed no evidence of neoplastic disease. Pathologic examination of the parietal pericardium, which measured 20 by 12 cm, revealed a chronic inflammatory process characterized by perivascular accumulations of lymphocytes. Some old hemorrhage and partially hyalinized fibrin were also present. The pathologic diagnosis was chronic fibrinous pericarditis.

Except for a small collection of fluid in the left pleural cavity, which subsequently disappeared, and a left basal pleuritis, the patient had an uneventful postoperative course. Immediately following operation, she was able to breathe much more comfortably and generally felt greatly improved, being discharged on the twentieth day after operation. The patient was last seen on Dec. 11, 1933, at which time she was feeling exceptionally well. She was

Fig. 2



Comment—This patient had dramatic relief immediately following parietal pericardiectomy for massive idiopathic pericarditis with effusion. A vigorous medical regimen, including many pericardicenteses, had failed previously to control the disease. The operation was technically easy at this stage.

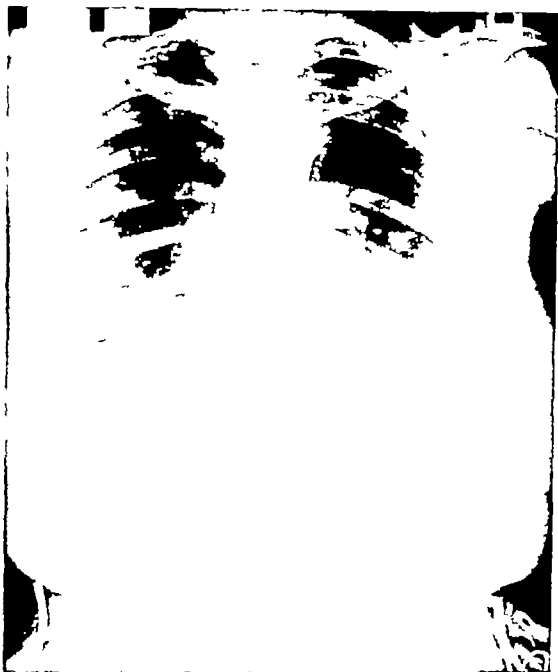


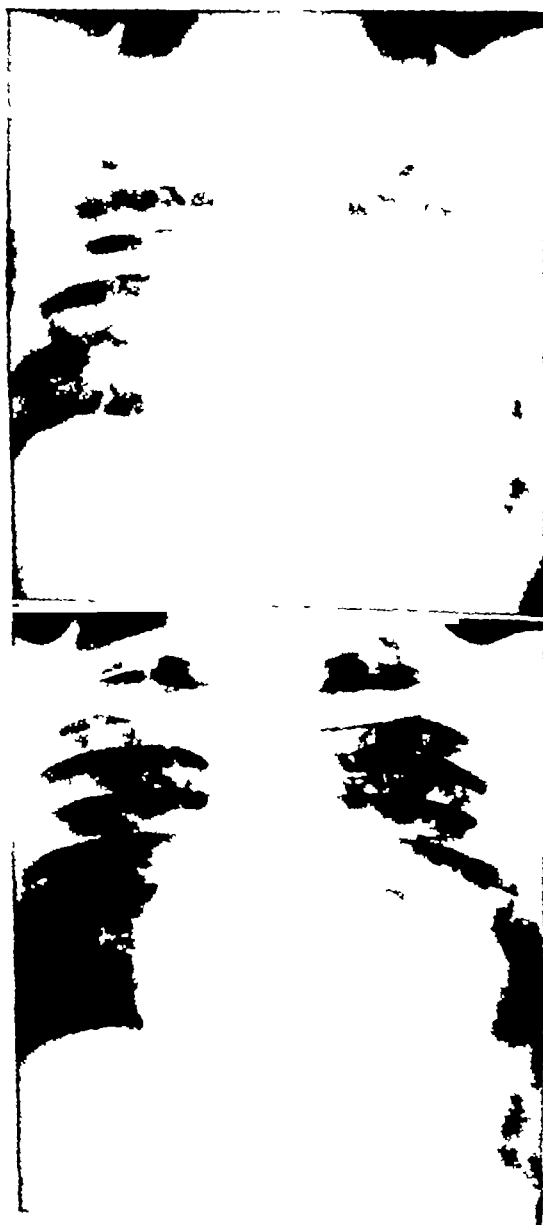
Fig 5—Case 2. A roentgenogram taken 18 months following an extensive parietal pericardiectomy. The heart and lungs are normal. There is some residual pleuritis at the left base.

CASE 3—H W, K C H. No 12732, a 48 year old Negro, was admitted to the Kings County Hospital on March 12, 1953. In January, 1953, the patient had developed a productive cough and had suffered from epigastric pain and anorexia relieved by the administration of antacids. In March, 1953, he had become short of breath while climbing only one flight of stairs. He had never been dyspneic before in his life, and there were no other cardiac symptoms. He had lost 10 pounds during the previous three months. There was no family history of pulmonary tuberculosis.

Examination revealed a middle aged Negro with a temperature of 103° F, pulse 100, blood pressure 118/80, and respiration 30. There were slight distention of the neck veins, dullness at the right base, and rales over the left lower lobe. The cardiac silhouette by percussion was enlarged to both the left and right sides, with frequent premature ventricular contractions. The liver edge was felt two fingerbreadths below the costal margin. There was no edema of the extremities. The venous pressure was 210 mm. of water, which increased to 310 mm. with pressure on the liver. The circulation time, with Decholin, was 30 seconds. The erythrocyte sedimentation rate was 24 mm and 60 mm. The blood count on two occasions and the urine analysis were normal. An electrocardiogram taken one week after admission showed low voltage in all standard leads with isoelectric to negative T waves. An investigation of the gastrointestinal and genitourinary tracts, including endoscopic and roentgen examinations, was negative for any abnormality. A parenchymal lesion was never detected in the roentgenogram of the chest, and the sputum studies were consistently nega-

tive for acid fast bacilli. Roentgen examination revealed a generalized and marked enlargement of the cardiac silhouette (Fig. 6). At fluoroscopy the cardiac pulsations were diminished. A tentative diagnosis of tuberculous pericarditis with effusion was made, and on March 20, 1952 the patient was started on a regimen of streptomycin and para-aminosalicylic acid.

FIG. 6



Within the next three weeks a total of four pericardicenteses was performed yielding a slightly turbid, yellow fluid. A smear of this fluid revealed the presence of acid fast bacilli. A total of 2,215 cc was removed from the pericardial sac on these occasions, the largest amount being obtained on April 9, 1953, when 1,200 cc was removed. After four weeks of antibiotic therapy and repeated pericardicenteses, the fluid was still forming, and the fever was essentially unchanged. Air had been instilled into the pericardial sac following one of the pericardicenteses, revealing a marked thickening of the parietal pericardium (Fig 7).

On April 21, 1953, operation was performed. An acute and chronic inflammatory process was found involving the parietal pericardium, which in some areas was as much as 2 cm in thickness. A few patches of fibrinous exudate were present on the visceral pericardium. These were easily removed. There was no evidence of constriction of the heart. An extensive parietal pericardiectomy was performed. The report of the pathologist indicated tuberculous pericarditis.



Fig 8—Case 3. A roentgenogram of the chest taken eight months following pericardiectomy. No abnormality is present, except for the elevated left hemidiaphragm.

Following operation, the fever gradually decreased, a normal temperature being reached on May 31, 1953, and being maintained thereafter. During this time the patient generally felt greatly improved, particularly in regard to dyspnea. By Sept 28, 1953, the patient had gained 7 pounds without evidence of edema. On Oct 6, 1953, the venous pressure was 70 mm of water, which did not change with pressure in the right upper quadrant. The Decholin time was 10 seconds. At the time of discharge on Dec 17, 1953, the patient was asymptomatic. A roentgenogram of the chest revealed only elevation of the left hemidiaphragm due to the removal of the phrenic nerve with the pericardium (Fig 8).

Comment—This patient had a pericardiectomy for tuberculous pericarditis as soon as the diagnosis was established, and a brief course of chemotherapy and antibiotics was administered before operation. Although the parietal pericardium was greatly thickened, it was easily removed. He has had an excellent result.

CASE 1—J. M. R. C. H. No. 50561, a 67-year-old Negro, was admitted to the Kings County Hospital for the first time on April 15, 1952, because of edema of the ankles, and marked dyspnea. Two pericardial aspirations yielded a total of 820 cc of fluid which contained neither red blood cells nor tumor cells. The patient was thought to have tuberculous pericarditis and a pericardiectomy was advised but he refused operation. During the next twenty to thirty months he was readmitted to the hospital on three occasions because of progressive orthopnea. He had received streptomycin, para-aminosalicylic acid, digitalis, Mercuhydrin, and a operation for ascites and pleural effusions. Despite this therapy his neck veins remained distended, a high venous pressure persisted measuring from 310 to 390 mm of saline fluid reaccumulated in the serous cavities, the liver remained greatly enlarged, and the cardiac silhouette gradually decreased in size. This course of events strongly suggested the development of constrictive pericarditis.

The 1:100 tuberculin test was strongly positive but repeated examination of the gastric juice, urine, pleural and pericardial fluid by smears and culture techniques did not reveal acid fast bacilli. The blood Kolmer was positive in 1:10 dilution. The basal metabolic rate was normal as were the following blood determinations: cholesterol, total proteins, albumin and globulin, urea, and glucose.

In January, 1954, orthopnea had progressed to the point where the patient was unable to sleep. The vital signs at that time were: blood pressure 110/70, pulse 80, respirations 28, temperature 99° F. He periodically developed a low grade fever ranging from 100° to 101° F. During the following month bilateral pleural effusions recurred following thoracentesis. The heart was not enlarged, its sounds were distant, and the rhythm was regular. The liver edge was five finger-breadths below the costal margin. The vital capacity was 1,200 cc. At *fluoro-graphy* very little pulsation of the cardiac borders could be detected. The electrocardiogram on four occasions was characterized by low voltage QRS complexes and inverted T wave, interpreted as evidence of myocardial damage.

Because of the severity of his symptoms and the need for frequent thoracenteses (Fig. 9), the patient finally agreed to operation which was performed on Feb. 4, 1954. Typical constrictive pericarditis was found, the heart being encased in fibrous tissue which was thick.

Over the right and left ventricles where it measured approximately 5 mm. The scar actually extended into the supradial layers of the myocardium necessitating a meticulous and difficult dissection. This was accomplished without incident, however, and following removal of the scar from the left and right ventricle and the base of the pulmonary artery, considerable improvement in cardiac action was noted.

The postoperative course was chronic fibrous pericarditis of undetermined etiology.

The patient had an uneventful postoperative course. Four months later when he was in the clinic his only complaint was occasional nocturnal dyspnea. His neck veins were not distended, there was no ankle edema, and the liver edge was palpable two finger-breadths below the costal margin, representing a considerable diminution in hepatomegaly. At the same time the chest roentgen revealed only residual pleural thickening (Fig. 10).

Comment. Although a good result was obtained in this 67-year-old man, we feel that the operation would have been easier to perform when it was originally recommended during the stage of effusion. Furthermore, the myocardial effects of two additional years of chronic pericarditis with cardiac compensation could have been avoided by early operation.

Fig 9

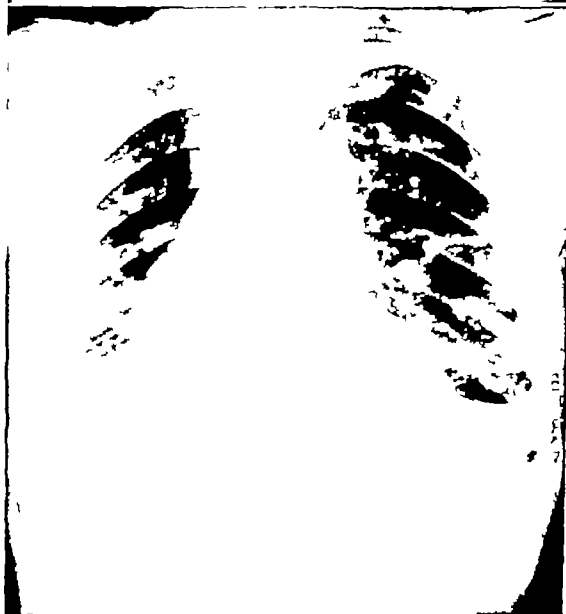


Fig 10

Fig 9—Case 4. A roentgenogram taken two days before operation illustrating the persistent right pleural effusion.

Fig 10—Case 4. Two months following pericardiectomy the only abnormality in the roentgenogram is bilateral pleural scars from the previous prolonged pleural effusions.

Following relief of cardiac silhouette depressed to a slight degree (Fig. 12). A kymogram taken at this time revealed pulsations of good amplitude despite the presence of fluid (Fig. 13). Marked ascites was present requiring the removal of as much as 6,500 cc of straw-colored fluid on one occasion. The venous pressure was 70 mm of saline, the circulation to the limbs was depressed, the neck veins were engorged, and the liver was enlarged 15 cm below the umbilicus. After repeated paracenteses the patient was discharged.

FIG. 11



Fig 13

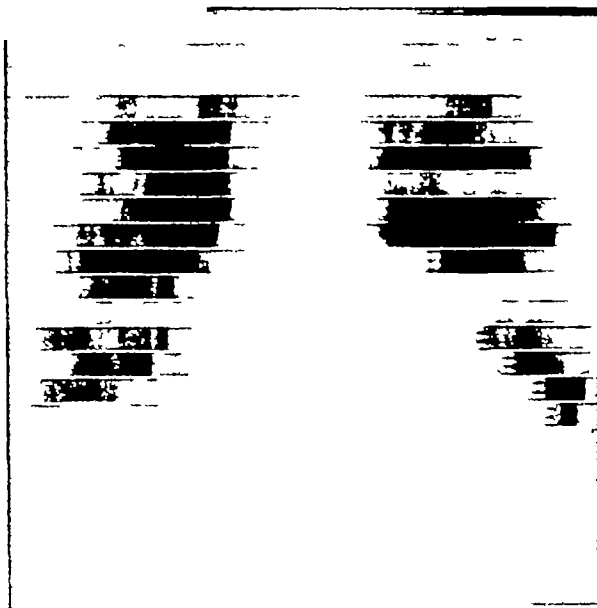


Fig 14

Fig 13—Case 5. A kymogram taken in 1949 during the stage of pericarditis with effusion. Note the peaked contours along the left lateral cardiac border indicating adequate diastolic filling and systolic ejection.

Fig 14—Case 5. In 1952 three years later the heart is normal in size but there is still fluid in the right costophrenic angle and in the oblique fissure.

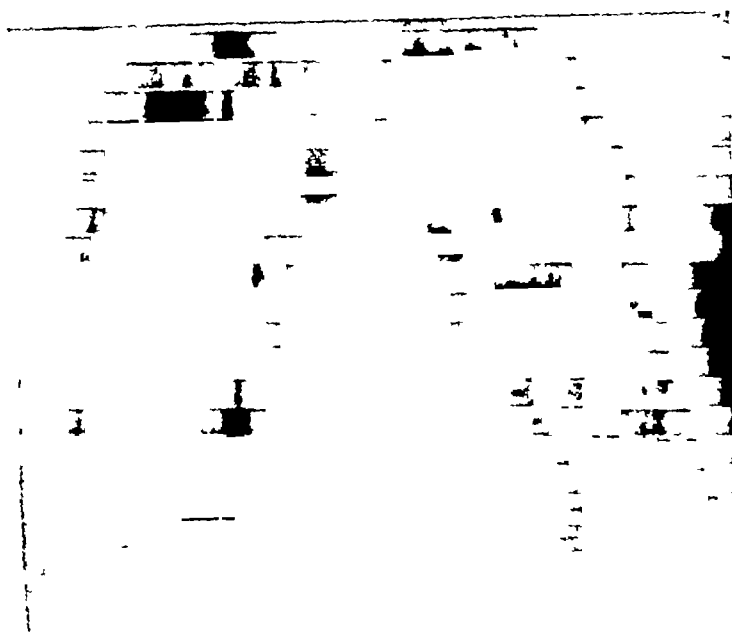
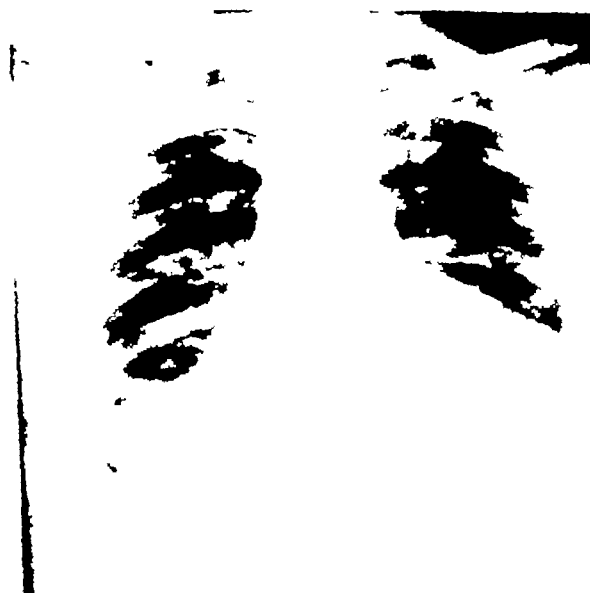


FIG. 1.—C. . . . A kymogram taken in 1954 during the stage of constrictive pericarditis. The superimposed fluorescent line along the left lateral cardiac border indicates the filling and systolic contraction of the ventricles.



somewhat improved. He was admitted for a second time on Jan 22, 1952, because of swelling of the abdomen, scrotum, and ankles. The total blood proteins were 5 Gm, the albumin was 3.3 and the globulin 1.7. Following abdominal paracenteses, he was discharged as improved six days later. It should be noted that although there were still signs of cardiac tamponade on this admission, the heart was now normal in size (Fig 14).

The most recent entry into the hospital was March 11, 1954, when he again complained of marked edema of the scrotum, penis, and abdomen. Physical examination revealed a temperature of 99.6° F, pulse was 80, respiration 20, and blood pressure 110/80. The neck veins were distended, the abdomen was markedly enlarged due to ascites, and an advanced degree of edema of the penis and scrotum was present. The venous pressure was 234 mm of saline, and the circulation time, with Decholin, was 20 seconds. The heart was small, and the kymogram was interpreted as evidence of constrictive pericarditis (Fig 15). A giant A wave in the jugular pulse tracings, faint heart sounds, and the previous existence of a pericardial effusion constituted substantiating evidence of this diagnosis.

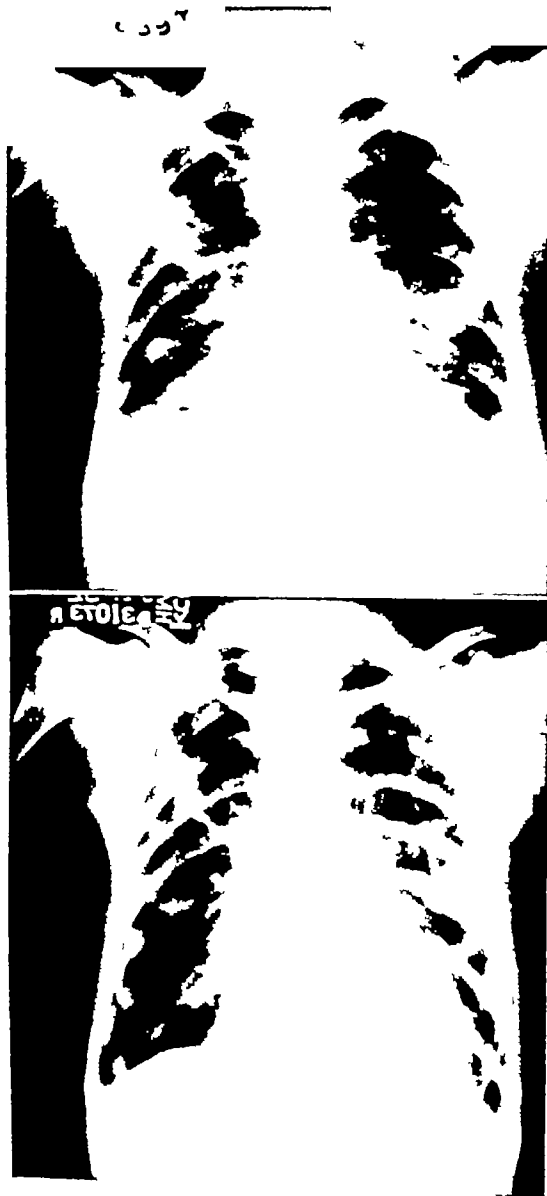
On a low salt diet, Mercuhydrin, and abdominal paracenteses, the patient's weight was reduced from 182 to 149 pounds within one month. On April 9, 1954, a pericardiectomy was performed. The operative findings included adhesions between the left lower lobe and the pericardium, and complete obliteration of the pericardial cavity by a constrictive pericarditis. The scarred pericardium measured about 4 mm in thickness, and after removal of it from the surface of the left and right ventricles and the base of the pulmonary artery, movement of the heart was greatly increased. During operation the heart was extremely irritable, becoming irregular and alternately slow and very rapid with the slightest manipulation. The patient withstood the operation very well, however, and had a relatively uncomplicated postoperative course. On May 10, 1954, he had ceased to accumulate peritoneal, pleural, and peripheral fluid and was feeling generally greatly improved. The pathologic report revealed acute and chronic nonspecific pericarditis. When examined two months after operation, the patient was asymptomatic. There was no ascites or edema of the ankles or distention of the neck veins. The roentgenogram of the chest was essentially normal (Fig 16).

Comment—This patient was treated conservatively for pericarditis with effusion. His symptoms persisted, and three years later signs of constriction were present but not recognized. The signs of advanced cardiac tamponade were eventually relieved by pericardiectomy performed five years after the pericardial effusion was first detected. It would appear, in retrospect, that an earlier operation was indicated.

CASE 6—T. McD., KCH No 10135-51, a 45 year old white man, was admitted to the Kings County Hospital on six occasions between 1949 and 1951 for congestive heart failure. On Dec 23, 1951, he complained of severe dyspnea and had recently had an arterial vascular occlusion of the right foot from which he had recovered spontaneously. He had received frequent mercurial injections and digitals.

Physical Examinations—The blood pressure was 120/88, pulse 88, respiration 20, and temperature 101° F. The lungs were clear. The heart was enlarged to the left anterior axillary line, and a gallop rhythm was present. There was no edema. The right foot was dusky and paler than the left. The blood count and sedimentation rates were normal. The circulation time, with Decholin, was 22 seconds. An electrocardiogram revealed low voltage QRS and T waves, with QS waves present in Leads II, III, and V₄. This was interpreted as indicating myocardial lesions and pericardial effusion. The roentgenogram of the chest revealed an infiltrate in the right second interspace with bilateral pleural effusion and a cardiac silhouette which was moderately enlarged (Fig 17). Tomograms of the heart revealed no calcific deposits. There were diminished pulsations along the left border of the heart by kymography and fluoroscopy. A second Decholin time was 23 seconds. A tuberculin test was positive in 1:10,000. The ballistocardiogram was normal. Blood sugar and urea were normal. Blood cultures were negative. The basal metabolic rate and measurement of

FIG. 17



I¹³¹ and creatine determinations were all within normal limits. The blood Wassermann test was negative. Numerous cultures of the sputum, urine, and gastric juice were negative for acid fast organisms.

The patient continued to have episodes of decompensation, engorgement of the neck veins, bilateral moist râles, enlargement of the liver, and dependent edema. Blood pressures persisted at about 90/80. A two week course of streptomycin and PAS was given, and on April 1, 1952, the patient was operated on under endotracheal anesthesia. The sternum was split and the pericardial cavity was found to be tensely filled with clear, watery fluid. No adhesions were present, but several small fibrotic patches were seen on the surface of the myocardium. The operative diagnosis was general myocardial degeneration. A large portion of the left anterior parietal pericardium was removed and the sternum was approximated. A thoracotomy drainage tube was inserted in the sixth interspace posteriorly. The pathologic diagnosis was subacute pericarditis.

The patient developed a superficial wound infection which healed satisfactorily. Eight weeks after operation the cardiac silhouette and the pulmonary infiltrate were smaller (Fig 18). It was noted that during convalescence, both in the hospital and later in the clinic, his dyspnea was greatly relieved. He felt much improved and had returned to work. Two years after operation he died unexpectedly at home. An autopsy was not performed.

Comment—It is not certain whether this man had pericarditis with effusion and associated myocardial degeneration with primary atypical pneumonia, as described by Godfrey,⁶ or whether the pericardial effusion was a manifestation of congestive heart failure due to idiopathic myocarditis. He was thought by the cardiologists to have the former syndrome. At operation, pericardial fluid was present under tension, and dyspnea was greatly relieved following a parietal pericardiectomy. This relief of symptoms continued until the time of his fatal attack.

DISCUSSION

We believe that the surgical treatment of patients with pericarditis should be formulated on the basis of a knowledge of the pathogenesis of the disease. Progression of pericarditis with effusion to the stage of constriction is not uncommon. Rigid distinctions between the stages of adhesive pericarditis (accretio cordis) and constrictive pericarditis (concretio cordis) may be misleading according to Moschowitz,⁸ who believes that adhesive pericarditis is simply the earliest stage of constriction.

If these concepts concerning the pathogenesis are valid, it would appear these are additional reasons why pericardiectomy should be performed in selected cases much sooner in the course of the disease, ideally during the stage of effusion. We recommend the early operation in both the tuberculous and idiopathic types of pericarditis whenever a prompt remission in constitutional symptoms and disappearance of the fluid do not occur with conservative therapy. Any patient with an appreciably thickened parietal pericardium in the presence of effusion should likewise have pericardiectomy. We have not employed this operation during the effusive stage of rheumatic pericarditis for fear of the presence of active carditis.

We prefer resection of the pericardium rather than pericardiostomy for the following reasons. (1) It provides better drainage of the pericardial fluid. It is well known⁹ that the pericardial sac, because of its configuration, is not

anatomically suited for complete dependent drainage (2) The problem of sealing over of the pericardial stoma is eliminated (3) Pericardiectomy removes a part or all of the focus of infection (4) Complete visualization of the visceral pericardium is obtained and resection of this layer can then be performed if such action seems appropriate to the surgeon

SUMMARY AND CONCLUSIONS

1 Six patients originally having pericarditis with effusion are reported In five patients the etiologic agent was undetermined, in one patient it was tuberculosis

2 All patients underwent pericardiectomy, four during the stage of effusion and two during the stage of constriction

3 All patients had signs of cardiac tamponade during the stage of effusion and at the time of operation

4 Pericardiectomy brought prompt relief of cardiac tamponade and striking general improvement in all patients One patient died unexpectedly two years after operation of undetermined causes after previously feeling well

5 Pericardiectomy is recommended during the stage of effusion in selected patients with tuberculous and idiopathic pericarditis Reasons for this have been discussed

We wish gratefully to acknowledge the counsel and assistance of Dr William Dock who has pointed out that the coronary arteries lying in the epicardium undergo narrowing or occlusion if the inflammatory process persists for a long time and that healing of the pericarditis may be followed by permanent vascular damage Dr Dock, therefore, has consistently urged early intervention in cases of chronic effusion with tamponade and tuberculous pericarditis with early evidence of cardiac embarrassment

REFERENCES

EXTRAPLEURAL PROSTHESIS CONCOMITANT WITH PULMONARY RESECTION FOR TUBERCULOSIS

PRELIMINARY REPORT OF 110 CASES

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AFTER a lobectomy, if preventive measures are not immediately taken, the space that has been created in the hemithorax is filled either by an adjustment of the remaining lung tissue or by deviation of the mediastinum. This results in an overdistention of lung tissue and presents two major hazards: first, reactivation of quiescent lesions through traumatic expansion, and second emphysema, with its consequent reduction of alveolar function. To prevent these complications such procedures have been advocated as immediate thoracoplasty, pneumothorax, pneumoperitoneum, and phrenic interruption.^{1, 2}

We have treated a series of 110 patients since December, 1950, by combining the lobectomy with a tailoring pleuroplasty followed by an extrapleural prosthesis. This experience leads us to believe that complications can be prevented fully as well by this method as by others, and that this method offers a wide range of benefits otherwise unobtainable.

INDICATIONS FOR PLEUROPLASTY

All the indications for performance of pleuroplasty are centered upon the problem of preventing overdistention of the ipsilateral or contralateral pulmonary tissues. The overdistention is prevented only if collapse is instituted immediately after the lobectomy. If, on the other hand, collapse is postponed and the remaining pulmonary tissue is allowed to expand for several weeks postoperatively, collapse will then be of little value. The harmful effects of overdistention will have already occurred.

Collapse concurrent with pulmonary resection is indicated by the presence of any one of the following conditions: (1) radiologic evidence of tuberculosis in the contralateral lung, (2) removal of the apical segments of the lower or middle lobe as well as removal of the upper lobe, (3) presence in the ipsilateral lung of many small, calciferous, and apparently quiescent lesions, (4) a lower lobe resection in which pneumoperitoneum cannot be instituted because of inflammatory changes, (5) inability of the remaining lobe, or lobes, to fill the hemithorax without overdistending. Inability is indicated: (a) when the lobes are extremely small but are free of palpable disease, (b) when multiple wedge or segmental resections of small lesions in the lower lobe have been performed, (c) when the remaining lobes show fibrosis from a burned-out tuberculosis, or (d) when the lungs are already emphysematous, a condition frequently found among older patients.

Protective collapse is not necessary when localized disease, although present, is without palpable involvement of the remaining lung tissue. Nor is collapse necessary when the resected lobe is small and is contracted and other lung tissue has already compensated. On the whole, however, we believe immediate collapse to be advisable. Jones and Robinson¹ for instance, have cited data which show that late complications are reduced when a thoracoplasty is performed concomitantly with resection.

ADVANTAGES OF CONCOMITANT LOBECTOMY AND EXTRAPLEURAL PROSTHESIS

In our experience extrapleural prosthesis performed at the time of the pulmonary resection shares the advantages but not the disadvantages of combined lobectomy and thoracoplasty. It is true that either combination provides effective protection against those complications that result from postoperative overdistention of the remaining lung tissue. Either combination accomplishes in a single operation what is usually done in two or more stages. However, when compared to the extrapleural prosthesis used in the present series, thoracoplasty represents a far more formidable procedure for the surgeon while for the patient it is more deforming.

Extrapleural prosthesis preserves the intact thoracic wall and maintains the wall's normal respiratory function. Paradoxical motion does not occur as it so often does after thoracoplasty.

Less blood is lost in extrapleural dissection than in thoracoplasty—a fact which assumes considerable importance when the lobectomy is performed concurrently.

The space left by the lobectomy can be filled more accurately by a tailoring pleuroplasty than it can be by a thoracoplasty.

Disturbance to the shoulder girdle is reduced by extrapleural prosthesis.

Because of the selective prosthetic collapse ventilation of the remaining ipsilateral lung tissue is more complete than ventilation after other methods of collapse—a matter of special importance during the immediate and the late postoperative periods.

The time required to perform an extrapleural dissection concomitantly with lobectomy is less than is required by the lobectomy-thoracoplasty operation.

Dissection with extrapleural prosthesis is a combination well suited to that of the older tuberculous patients in whom the clinical picture is complicated by pleural adhesions. By virtue of its shorter operating time, shorter anesthetic and blood loss the combined operation is also more suitable for older patients.

Combining these procedures into one reduces two operation morbidities.

Sooner recovery is obtained by the combined procedure, financial ease to the patient.

dicates the necessity of concomitant collapse and extrapleural prosthesis, the usual sequence is to proceed with the intrapleural freeing of the lung, accomplish the pulmonary resection, and then fashion the extrapleural pocket to fit the space

On the other hand, development of the extrapleural pocket is done before the lobectomy, whenever adhesions over the upper lobe are so dense there is a danger of fragmenting diseased lung tissue near the pleural plane. This alternate sequence, incidentally, facilitates separation of the parietal and visceral pleura since it brings the apex of the lobe down into view where a more meticulous dissection can be carried out.

Intact preservation of the pleural layers is not essential. Where there is danger of entering the lung, the surgeon need not hesitate to remove localized areas of both of the pleural layers. As a matter of fact, in some patients of our series the floor of the extrapleural pocket had as many holes as Swiss cheese. Such conditions make little difference in the end result provided the pleura forms a confining web which will retain the prosthetic spheres.

After the extrapleural pocket has been fashioned, any large spaces inadvertently made in the pleural diaphragm are closed, but smaller openings are allowed to remain for drainage. Intrapleural catheter drainage was found adequate for the clearing of both intrapleural and extrapleural spaces.

REVIEW OF CASES

The 110 patients in the series ranged in age from 7 to 65 years. A gradual trend was noted of increasing age, the resections during the period being performed on increasingly older patients. Nine per cent of the patients were over 50 years of age (Table I).

TABLE I SEX AND AGE DISTRIBUTION OF CASES

AGE IN YEARS	CASES
7-20	10
21-30	26
31-40	39
41-50	24
51 and over	11
Male	52
Female	58

In most instances, resection was limited to the upper lobe but the series included a variety of lobal segments. For instance, in two cases of lower lobectomy, the prosthesis was localized in the costophrenic sulcus (Table II).

The size of the prosthesis was, of course, governed by the volume of lung tissue removed. The average prosthesis consisted of six spheres, each of which measured $1\frac{1}{4}$ inches in diameter and occupied approximately 1 cubic inch of space.

Three types of prosthesis were used. The first 17 patients in the series had an air space, developed at the time of resection, which later was converted to an oleoplombage. In one case a Lucite sponge was used. Lucite spheres were employed in the last 92 cases.

TABLE II EXTENT OF RESECTION

	NO OF CASES
Right upper lobe	47
Left upper lobe	33
Right upper lobe and superior segment of right lower lobe	6
Left upper lobe and superior segment of left lower lobe	3
Right upper lobe and decortication of right lower lobe	2
Left upper lobe and decortication of left lower lobe	1
Right upper lobe and wedge of right lower lobe	3
Left upper lobe and wedge of left lower lobe	4
Right upper lobe and right middle lobe	3
Posterosuperior and anterior segments of left upper lobe	3
Superior segment of left lower lobe	1
Posterolateral segment of left upper lobe and superior segment of left lower lobe	2
Right lower lobe	1
Right lower lobe and wedge of right upper lobe	1

We encountered none of the major complications from prosthetic replacement that are given in some of the reports. Neither effusion nor migration of prosthetic material was encountered. Infection of the prosthetic space—except in association with bronchopleural fistulas—was encountered only in the one case of prosthesis by Avalon sponge. Complications that were met in the series are given in Table III.

TABLE III PROSTHESES AND COMPLICATIONS

TYPE OF PROSTHESIS	NUMBER OF PATIENTS	PATIENTS WITH COMPLICATIONS	COMPLICATION	DEATHS
Avalon sponge	17	2	Bronchopleural fistula	2
Decortication	1	1	Infection of space	0
Left upper lobe	6	6	Bronchopleural fistula	1
			(3)	0
Total	110	6	Spread of disease (3)	3

The five infections encountered in the series resulted from bronchopleural fistulas and these would probably have occurred with or without prosthesis. In four of these five cases the disease was far advanced and bilateral. All five were difficult resections. Two required lobectomy plus decortication and had many small bronchopleural fistulas present on closure. Two others required segmental resections of the superior division of the lower lobe in addition to upper lobectomies. The fifth case was technically a relatively easy case in which standard plombage was used, the patient developing the fistula two and one half years later.

Two of the patients in the series

Spread of disease followed surgery in three patients of the series. One patient, a Mexican teen-age girl, was in the desperate risk classification preoperatively. Sixteen months after a right upper lobectomy, she still is in the sanatorium, slowly improving, but still sputum positive. The remaining two patients who suffered spread of disease were recalcitrant to postoperative therapy. Neither accepted bed rest and one refused chemotherapy.

Infection of the prosthetic space, without bronchopleural fistula, occurred in the one patient in whom a Lucite sponge was used as the prosthetic material. Since the dominant growth of the infection was *Pseudomonas aeruginosa*, we suspected contamination of the space as a result of difficulties in sterilizing the prosthetic material, and we abandoned further use of sponge prosthesis.

Several causes account for the three deaths encountered in the series. The first death occurred in a male patient who was discharged from the sanatorium as a probable cure. Six months later he was found dead in his home. Death had taken place several days previously, and the cause has never been determined. The second death happened four days after resection and no autopsy was obtained. Cause of death was assumed to be a pulmonary embolus. The third death in the series was the Oriental patient mentioned in the discussion of bronchopleural fistulas.

All of the complications mentioned previously occurred in the first 48 patients in the series, whereas the last 62 patients were free of these difficulties. As will be seen, this improvement resulted, in all likelihood, from the change of the prosthetic material from oil to spheres, and from the discovery of a more adaptable method of using these spheres.

PROSTHETIC TECHNIQUE

Our technique for extrapleural prosthesis has evolved through several developmental stages. Our first patients in the series were given a temporary air space which later was filled with oil. While this method fulfilled a few of the criteria for good functional prosthesis, an airtight pocket could not always be obtained at the time the operation was performed since the parietal pleura was occasionally torn in the process of separating adhesions. This, and the need for periodic pressure checks—and, in several patients, the loss of the air space—were factors which combined to force our search for a more satisfactory prosthesis.

In one case in which an airtight pocket was unobtainable a temporary substitute was fashioned with a Brake bag. The result was very satisfactory in maintaining the pocket, but we had difficulty later in removing the bag, and we felt also that the hazard of infection was too great to warrant a continuation of this procedure.

Gale and his associates³ have reported the use of Lucite sponge for extrapleural prosthesis, our experience with an infection following the use of an Avalon sponge prompted our change to the use of hollow Lucite spheres.

Spheres were used in 92 cases. In the first of this particular series, we tied the spheres together and secured the resulting cluster to Sibson's fascia

FIG. 1



Fig 3



Fig 4

Fig 3—R. A. a 44 year old man with bilateral apical tuberculosis accentuated on the right side. Preoperative bronchspirometry studies revealed borderline pulmonary reserve due to bilateral lower lobe emphysema. The preoperative studies indicated that further distention of emphysemic areas would result in further diminution of borderline alveolar function.

Fig 4—Same patient following right upper lobectomy with replacement extrapleural prosthesis.



Fig. 5. Intraoperative film of extrapleural Tulle prostheses following left upper lobectomy.



Several methods of grouping the spheres were tried. For instance, holes were drilled through their centers for ligature grouping. In every case of this kind, levels of fluid developed inside the spheres, a matter which not only caused confusion in x-ray follow-up procedures but which turned the spheres into potential foci of infection.

As a means of keeping the spheres together, small orthopedic screws were inserted in their walls and the ligatures attached to these. Solid balls of the Lucite with holes bored through them were also tried in our search for the best prosthetic method. These last two maneuvers were effective, but we later found it unnecessary to group the spheres at all.

In each of the final 85 patients in the series the spheres were emplaced loosely and were localized only by the reconstituted pleural diaphragm. This method of extrapleural prosthesis has proved to be the most adaptable and has resulted in fewer complications.

COMMENT

Postoperative complications which result from overdistention of the remaining lung tissue are reduced whenever collapse is instituted concurrently with the lobectomy. Pulmonary resection in this manner and replacement by a prosthesis is, of course, attended by the same complications either procedure entails when done separately. Immediate postoperative difficulties consist of operative shock and bronchial spread of the disease. Later complications may embody the various stages of infection, including bronchopleural fistula, development of empyema, and spread of disease by overdistention of quiescent lesions. However, in the 110 cases discussed, complications occurred in only 8 per cent, while the final 62 cases (56 per cent) were free of complications.

The ratio of complications to cases was approximately 1 to 15 in the sphere group as compared to 1 to 8 in the oleoplombage group.

Since, proportionally, the large series of cases in which Lucite spheres were used had about 50 per cent fewer patients with complications than had the small earlier prosthetic group, it may be concluded that loosely emplaced Lucite spheres offer a far preferable prosthetic method.

SUMMARY

1. Extrapleural prosthesis was performed concomitantly with resection in a series of 110 cases. Prosthesis consisted of oleoplombage in 17 cases, of a Lucite sponge in one case, and of Lucite spheres in 92 cases.

2. The indications for preventive collapse following resection are given.

3. Concomitant lobectomy and tailoring pleuroplasty for extrapleural prosthesis offers surgeons and patient a number of advantages over the combination of lobectomy and thoracoplasty.

4. The technique is outlined for the surgical procedure, emphasis being given the problem of developing the extrapleural pocket.

5. Complications occurred in only 8 per cent of the cases. The complications were bronchopleural fistula, spread of the disease, and infection of the prosthetic space.

6. Three deaths occurred in the series. One was six months after discharge and was of unknown cause. One was presumably caused by a pulmonary embolus. One followed an overwhelming infection that accompanied a broncho-pleural fistula.

7. Various types of extrapleural prosthesis were tried. Free Lucite spheres placed above a reconstructed pleural diaphragm were most satisfactory.

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SUGGESTED MODIFICATIONS IN THE APPLICATION OF THE SIGMAMOTOR PUMP TO THE TECHNIQUE OF CONTROLLED CROSS CIRCULATION

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FAMILIARIZATION experiments in dogs using the controlled cross circulation technique of Lillihei and associates^{1 2} for the direct vision method of closure of certain congenital intracardiac defects in human beings have led to several valuable modifications in the application of the Sigmamotor Pump

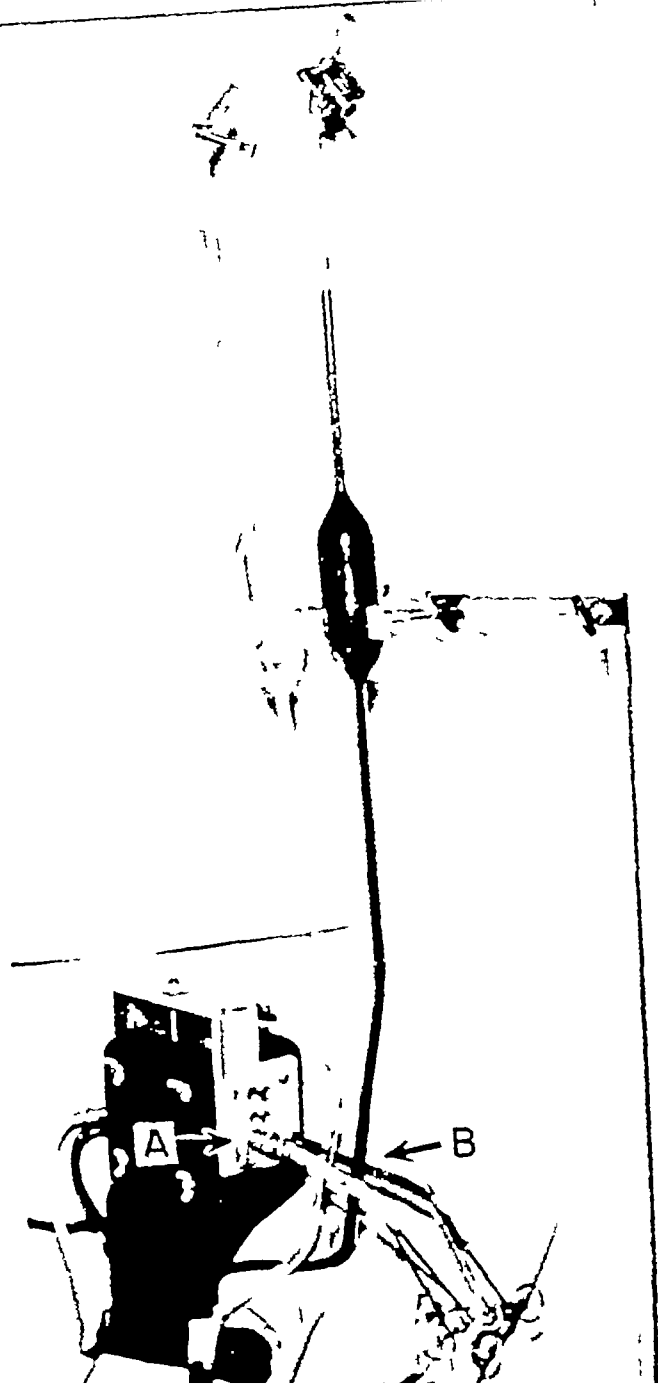
By means of the multifingered Sigmamotor Pump, which massages fluid by serially applied pressures on the outside of two pieces of tubing, equal rates of pulsatile blood flow may be established in two external circuits of small volume. In use, the circuits are connected (Fig 1) by means of catheters so that oxygenated blood from a donor artery is pumped into the ascending aorta of the recipient, and the venous return, excluded from the right heart, is collected from the superior and inferior venae cavae of the recipient and pumped into the venous system of the donor. If the flow rates are equal, blood volumes in the donor and recipient remain unchanged, and the donor, through hyperventilation with 100 per cent oxygen, is capable of aerating both his own circulation and that supplied to the recipient.

Some difficulties inherent in this procedure have been met by the following modifications:

- 1 An adjustable collar for fine calibration of flow balance
- 2 A reservoir applied to each system as a hydraulic cushion in the event of restriction of either inflow catheter
- 3 A means of rapid replacement of blood loss during the operative procedure
- 4 Plastic ridges applied to the caval catheter to facilitate placement through the heart

METHOD

These modifications are illustrated in Figs 1 and 2. A short piece of Tygon tubing is cut so that when securely adapted over glass connecting tubes, with the closed adjusting collar against the input face, a slight amount of tension is maintained on the Tygon when mounted in the pump. An inverted 100 ml pipette is connected in each system to the upright stem of the glass crossarm with a short piece of Tygon, and the tip of the pipette is closed with a rubber tube and clamp. The side arm is connected to a small diameter



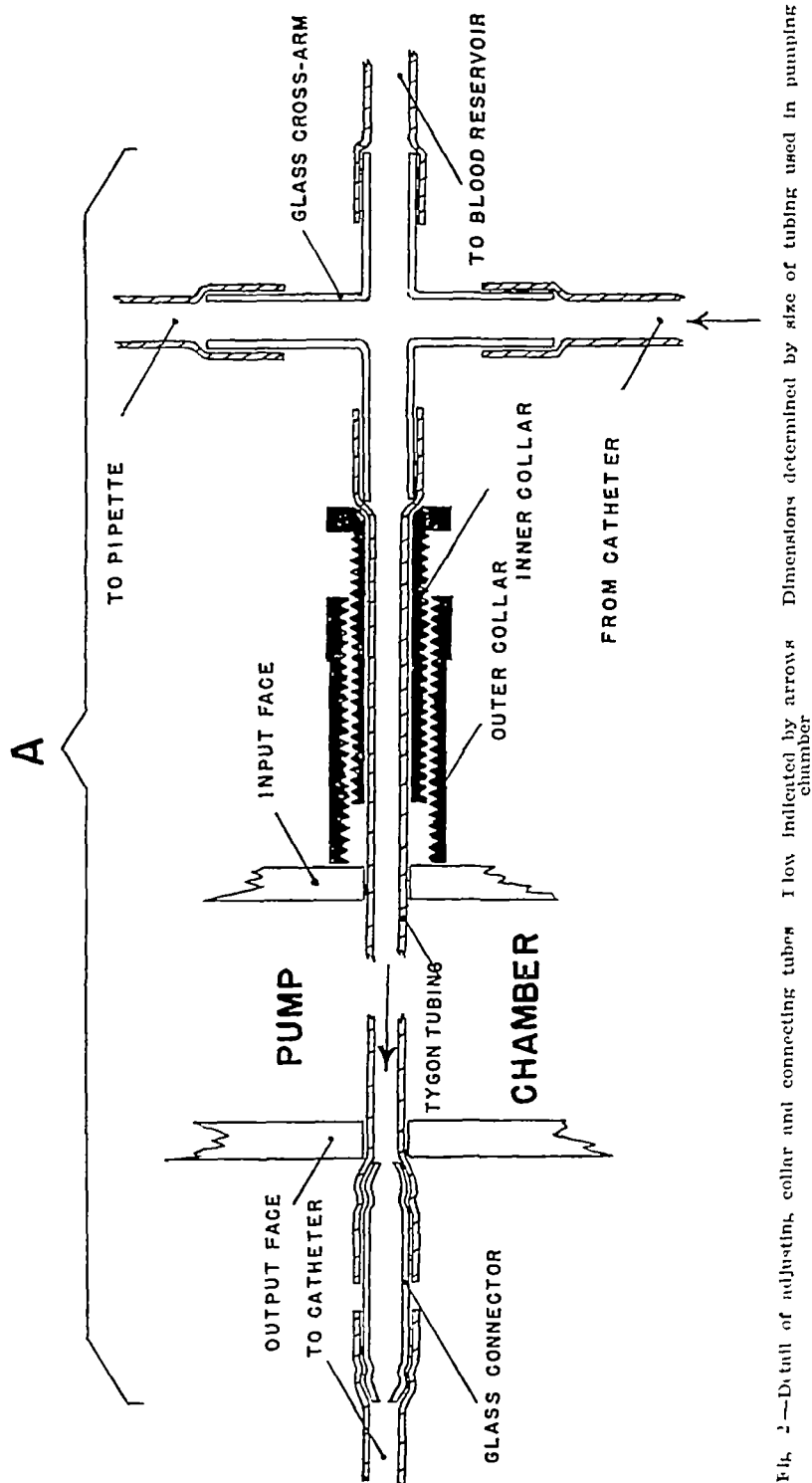


Fig. 2.—Detail of adjusting collar and connecting tubes. Flow indicated by arrows. Chamber

Tygon tube which is clamped ready for use with an infusion setup. The system is filled with saline by running the pump, a valved suction bulb being used to draw saline into the pipette. The system is freed from bubbles and the fluid reservoir is partially degassed by repeated clamping of the intake catheter while the pump is operating.

DISCUSSION

Sensitive flow balance within 1 cc/5 min. may be made with an adjustable control collar on each circuit. By distracting the control collars, it is possible to decrease the internal diameter of the tubing passing through the pump, thereby obtaining a decrease in output volume per pulse. The glass connectors fitting the tubing tightly are held at the outlet face and at the end of the control collar to eliminate creep of the tubing through the pumping chamber.

The level of the saline in the pipettes is a sensitive indicator of flow dynamics; any restriction of intake resulting in a sharp lowering of the fluid level in the narrow upper stem. Since such restriction of intake will often suck air into the circuit at the point of adaptation of tubing and catheter or at least will cause extensive degassing of the blood, the presence of the hydraulic cushion is a source of fluid supply until the catheterized vessel can refill. A standard Abbott disposable Blood Recipient Set is used as an elective supplementary blood supply either to augment blood volume in the event that an intake catheter becomes occluded because of insufficient supply or to compensate for coronary spillage while the heart is open. The needle adapter is connected directly to the side arm and blood is drawn into the circuit when ever the side arm clamp is released.

Bubbles in the circuit may be persuaded to find their way into the pipettes by proper manipulation of tubing and catheters eliminating disconnections and/or the catheters are inserted.

pressure head in the donor individual and through obstruction of the venae caval catheter in the recipient individual. The latter complication is frequently initiated by excessive arterial blood loss through an incompetent aortic valve and the ventricular septal defect in the open heart. The venous return is thereby diminished and the venae cavae are sucked into the intake openings of the catheter. This chain of events can be controlled to a great extent by intermittent occlusion of the ascending aorta while the heart is open, thereby preventing regurgitation of blood through the aortic valve. In addition, smaller intake holes in the collecting catheter, or the incorporation of a blood reservoir between the recipient individual and the pump, will reduce the hazard of catheter obstruction in the recipient individual. We feel, however, that it would be beneficial to have an efficient, but not too complicated flow-meter incorporated in each circuit so that the exact rates of blood flow in both directions would be known at any given instant.

SUMMARY

Modifications are suggested for the use of the Sigmamotor Pump in cross circulation procedures. They present the following improvements and safeguards:

- 1 Accurate and permanent adjustment of flow balance by means of a simple expansion collar
- 2 Diminution of the major hazard of an embolus from the extracorporeal circuit by furnishing a fluid buffer system
- 3 A readily observable indicator of flow dynamics
- 4 Addition of a blood reservoir for rapid replacement of blood volume as necessary
- 5 A method aiding the accurate and secure placement of the catheter in the superior and inferior venae cavae of the recipient

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A NEW FINGERNAIL COMMISSUROTOMY KNIFE FOR MITRAL VALVE SURGERY

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WE WISH to report the development of a new fingernail mitral commissurotomy knife which greatly simplifies the technique of mitral commissurotomy. Concomitantly with this instrument the safety of the procedure is enhanced inasmuch as the inadvertent production of a complicating mitral regurgitation is obviated.

The pathology of the stenotic mitral valve has been described at length^{1, 2} and is not a topic for discussion in this paper. It is sufficient to say that the end result is a narrowed slitlike rigid valve orifice with thickened valve leaflets, and with agglutination and thickening of the chordae tendineae. The chordae are aligned longitudinally in two rows, one to the edge of each agglutinated valve leaflet. The anterior commissure of the stenotic mitral valve tends to form a fine curved grooved line extending from the anterior portion of the stenotic orifice to the myocardial wall. This fine line is the landmark which the surgeon uses to guide safely the splitting of the stenotic valve between the chordae. If the valve should be split outside of this line, a serious complicating mitral regurgitation would result. Splitting the valve outside the line in the direction of the mural leaflet is not attended by nearly as significant a regurgitation as would be produced by a similar split in the aortic leaflet. Also the instrumental cutting or the digital tearing of a chorda tendinea is attended by a flaccid valve leaflet and a severe regurgitation.

Not every stenotic mitral valve requires instrumentation during the performance of mitral commissurotomy. An instrument is required in about 50 per cent of the cases, however, if the commissure is to be completely opened to the heart wall. A few valves split easily by mere insertion of a finger through the orifice. Calcified valves usually split easily initially after the finger has been inserted. They do not always open easily to the heart wall, however, and may require additional instrumentation to obtain an adequate two-finger opening. The valves which invariably require cutting are those which theoretically should give the best results, and yet are fraught with the greatest danger of a complicating regurgitation. These are the slightly thickened non-calcified valves which are freely mobile save for the presence of a thick leaflet or commissure around the stenosed orifice. These valves do not split readily by digital commissurotomy and invariably require mitral instrumentation. As soon as the finger enters the annulus the mitral valve splits easily on application of

pressure with the tip of the finger or further instrumentation may be required to gain an adequate two-finger opening. These are the valve leaflets which are almost normal and theoretically, should be attended by the best long-range functional results. Yet, a slight deviation in the cut from the true commissural line may result in a regurgitation which is at least as threatening as the original stenosis if not more so.

At all times it must be remembered that it is of utmost importance to perform the commissurotomy without the production of a regurgitation or without significantly increasing any minor existing one. In the accomplishment of this the integrity of the commissure must not be violated.

Several factors of importance must be considered in the designing of an instrument:

- 1 The instrument must facilitate extremely delicate cutting of the agglutinated valve leaflets. It must make possible meticulous commissurotomy and permit small incisions so that following each cut a regurgitant jet may be felt for by means of which an early minimal regurgitation can be detected. It should not permit the performance of a gross split, which may produce a large regurgitation. The production of a significant regurgitation may very well prove to be fatal or may produce congestive failure in the postoperative period.

- 2 Frequently, commissurotomy must be performed in the presence of a thrombosed auricular appendage or an intra-atrial thrombus. Under this condition gentleness in technique is of utmost importance in the prevention of manipulative embolization. It is important to design an instrument which can be worn upon the initial entrance of the finger into the atrium and does not necessitate repeated reinsertions of either the finger or other instruments into the heart. Obviously each manipulation enhances the risk of embolization.

- 3 The instrument must be one which does not impair full tactile sensation by the delicate palpatory tip of the index finger. Simultaneously, it must facilitate the accurate cutting of the stenotic valve exactly along the commissural line.

- 4 It should be one which permits the index finger to palpate the valve leaflets and the commissure at the identical moment that it is performing the instrumental commissurotomy.

- 5 It must be an instrument which is carried on the index finger at the time of its initial entrance into the left atrium and may be used or not at the discretion of the surgeon. In the event that the knife is not needed it must not in any way hinder digital action during the commissurotomy.

Several types of instruments have been previously devised for the performance of instrumental commissurotomy.^{3,9} Some are introduced into the left atrium along the volar aspect of the index finger and prevent simultaneous palpation of the valve at the exact time of instrumentation, when it is needed most. Others are worn over the tip of the index finger. These require several

insertions of the finger into the heart and by covering the tip of the finger, preclude the possibility of its adequate palpatory sensation during the cutting of the valve. An inaccurate cut will be attended by the usual complications of a poorly aligned commissurotomy, namely, regurgitation congestive failure, and possible subsequent mortality.

After considerable experience in the palpation of stenotic mitral valves and in the performance of mitral commissurotomy, one of us (A. A. B.) noted that when the valve annulus was fibrotic or calcific and difficult to open, he always attempted to split the anterior commissure with the fingernail of the index finger. This experience provided the initial stimulus and realization of the necessity for the development of our present knife.

It was obvious that it was necessary to develop a steel fingernail knife one which in effect replaced the free edge of the existing nail by a small steel blade. Concomitantly, it was essential to preserve the integrity of the palpatory tip and side of the finger so that it was in no way impeded during the palpatory exploration of the mitral valve and the left atrial chamber.

The knife which we have developed is a fingernail commissurotomy knife. Its use of a narrow snug fitting ring placed high around the distal phalanx, a steel fingernail is maintained in position over the existing fingernail which, in turn, places a small crescent shaped blade immediately beneath the free edge of the nail (Fig. 1). This blade is sharply edged and projects beyond



the perionychium. This serves to conceal the entire knife within the contours of the existing nail. A small hole is made in the ring portion for the attachment of a length of steel wire, as an added safety factor in preventing its disengagement while in the heart.

The entire knife is made of stainless steel. There are no sharp edges or corners to cause snagging while in the heart, and simultaneously, there are no inaccessible surfaces to prevent its easy cleaning and sterilization. It is best sterilized in ordinary surgical scalpel sterilizing solution.

Fig 2



Fig 3

Fig 2—Oblique view of the right index finger showing the fingernail knife in place on the distal phalanx. The crescent-shaped blade is beneath the edge of the fingernail and is maintained in position by it.

Fig 3—Close up of Fig 2 showing the fingernail knife in place. The palpatory surfaces of the finger tip and the side of the finger are not impaired by the instrument.

The knife may be used on the bare finger, as some surgeons prefer to enter the heart, or it may be used under a rubber glove (Fig 5). Should a glove be worn after it is slipped on over the knife a small piece of rubber over the very tip of the index finger must be excised so that the knife blade itself may be properly bared while performing the commissurotomy.

Because of the tremendous variation in the size, shape, and contour of the distal phalanx and nail of the index finger of different individuals it be

is obvious that although it might be possible to establish standard sizes, in order to really fit perfectly and work well the knife was best suited to be individually custom made. Consequently the manufacturer of the instrument has provided for individualization in the fitting and casting of the fingernail commissurotomy knife. This may be performed with only a minimal amount

FIG. 4



SUMMARY

1 A custom-made fingernail knife has been developed and described in detail

2 This knife enhances the safety of the operative procedure of mitral commissurotomy in facilitating the accurate cutting of the stenotic valve exactly along the commissural line

3 The precision with which the commissure can be cut obviates the production of any significant mitral regurgitation

4 The instrument facilitates delicate cutting of the stenotic mitral valve

5 The knife is worn on the index finger during its initial entrance into the heart, which obviates the reintroduction of the finger and other instruments. This is of importance in the prevention of embolization in the presence of auricular and atrial thrombi

6 The instrument does not impair the palpatory ability of the finger tip. It may or may not be used, at the surgeon's discretion. If it is not used, it in no way hampers digital commissurotomy

7 The knife is hidden in the recesses of the contour of the perionychium. This serves to sheath the blade of the knife so that its cutting action can be employed only at the surgeon's discretion

8 Most important, it permits the valve leaflets and commissure to be palpated while the instrumental commissurotomy is being performed. This is the moment when simultaneous palpation is most crucial

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USE OF THE RECORDING OXIMETER IN MANAGEMENT OF POSTOPERATIVE OXYGEN THERAPY

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PATIENTS who have had a surgical procedure involving the chest are found to be in a state of lowered arterial oxygen saturation in the immediate post-operative period. This can be explained on the basis of poor ventilatory exchange due to central depression from anesthetic agents, incisional chest pain and retained secretions, producing physiologic blood shunting through poorly or nonventilated areas of the lung. In young patients and in the absence of cardiorespiratory complications there is a spontaneous tendency to return to normal levels of arterial oxygen saturation shortly after surgery when consciousness is complete. Mild sedation is then administered and cough is encouraged. In older patients the return to normal levels of arterial oxygen saturation is slower, cardiorespiratory complications appear more frequently and functional adaptation to the new situation created by surgery takes longer.

Many authors have contributed a number of well-established facts to our knowledge and application of oxygen as a therapeutic agent. Cardiopulmonary physiologists like Courmand, Comroe, Christie, Wright and many others have devoted numerous publications to such problems. Barach in his book on oxygen therapy made a very complete and careful exposition on the matter. A recent publication by Andrews deals with this subject.

The deleterious effect of hypoxia has been repeatedly demonstrated and should be prevented or corrected by all available means in a patient who has just undergone major surgery and has still to overcome the injuries inflicted by the anesthetic. Since the conditions primarily responsible for this transient hypoxia cannot be directly acted upon for its prevention, the routine practice of increasing the oxygen content of the gas mixture breathed by the patient during the postoperative period is effective in relieving this hypoxia. It should be continued until the patient is able to maintain an adequate

In the past, changes in pulse, respiratory rate, and clinical condition of the patient were used in common clinical problems as criteria to institute, stop, or regulate the amount of oxygen needed by the patient. It was then considered advisable to administer the patient oxygen flows between 5 and 10 L per minute via a nasal catheter, and any flow under 5 L per minute was thought to be of little value to the patient. These data, when properly interpreted, are of considerable help, but they do not have the same direct measuring quality as the arterial oxygen saturation level in determining the oxygen requirements of an individual case.

The use of the continuous-recording oximeter for the study of the clinical problems in this institution furnished us with an accurate method of judging the effectiveness and indications of the different techniques of oxygen therapy applied in the routine postoperative treatment of surgical chest cases and to correlate the validity of clinical criteria.

METHODS

Fifty patients on whom different thoracic surgical procedures were performed furnished the clinical material used in this study. Among the surgical procedures were pneumonectomies, lobectomies, segmental resections, thoracic plasties, aortic resection, mitral valvulotomies, and esophageal surgery. All these patients had reduced ventilatory efficiency, partially due to an operation on the chest wall.

Those with pulmonary resection had the additional factor of a reduction in lung capacity (Table I). A continuous recording, Wood type, earpiece oximeter, built at The University of Chicago by Dr. Perkins and associates, was used for the arterial oxygen saturation determinations. The recording paper speed was set at 1 cm per 2.5 minutes in order to make it suitable for several hours of continuous recording (Fig. 1).

TABLE I. USE OF THE RECORDING OXIMETER IN MANAGEMENT OF POSTOPERATIVE OXYGEN THERAPY

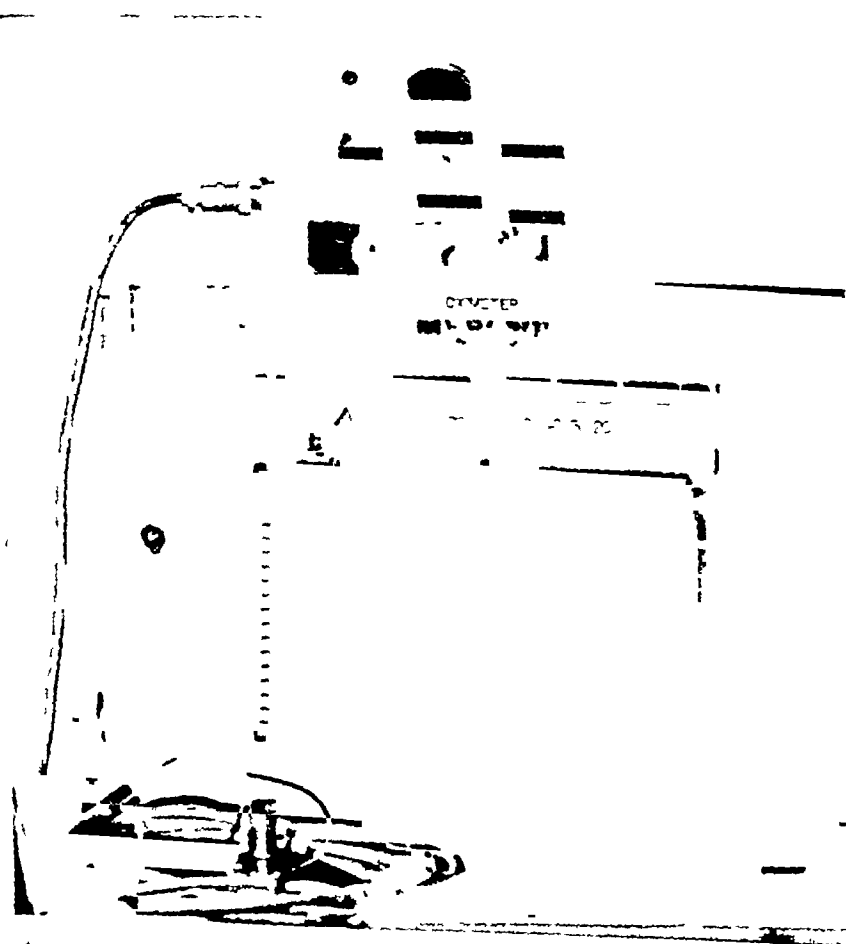
DISTRIBUTION OF OPERATIVE PROCEDURES	
Pneumonectomy	17
Lobectomy (partial or complete)	16
Exc. of emphysematous blebs and bullae	1
Chest wall operations	5
Miscellaneous intrathoracic (without excision of lung)	11
Total	50

Immediately after surgery the patient was transferred to the recovery room, where pulse rate, respiratory rate, arterial oxygen saturation, and clinical condition were carefully recorded on the oximeter record.

A base line for arterial oxygen saturation, pulse rate and respiratory rate was then established by allowing the patient to breathe room air long enough to get a plateau for at least five minutes.

Oxygen, 100 per cent, was then administered by the following means: (1) a face mask connected to a McKesson machine with positive pressures of 10

mm Hg applied (2) B I B face mask with an oxygen flow of 10 to 12 L per minute (3) via a nasal catheter as soon as the patient regained consciousness. Previous to this time the patient's tidal volume was insufficient to allow the use of a nasal catheter. Flows of 1, 2.5, 5, 7.5, and 10 L per minute were then established, sufficient time being allowed between flow changes for the patient to reach sustained levels of saturation. The efficiency of the oxygen therapy was judged primarily on the basis of arterial oxygen saturation values; the patient was left on the lowest oxygen flow per minute necessary to maintain an adequate arterial oxygen saturation.



RESULTS

In this series, 90 per cent of the patients showed a decrease in arterial oxygen saturation following surgery lasting from three hours to several weeks after the operation, depending primarily on the presence of cardiorespiratory complications. The average volume of all patients was 88 per cent of arterial oxygen saturation twenty-four hours after the surgical procedure, whereas normal figures of arterial oxygen saturation range from 96 to 98 per cent.

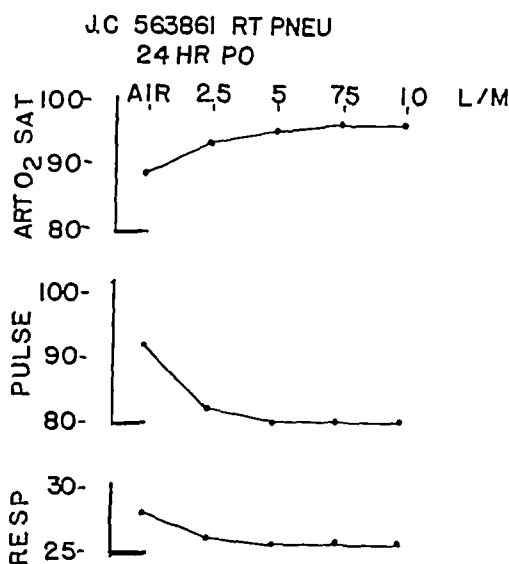


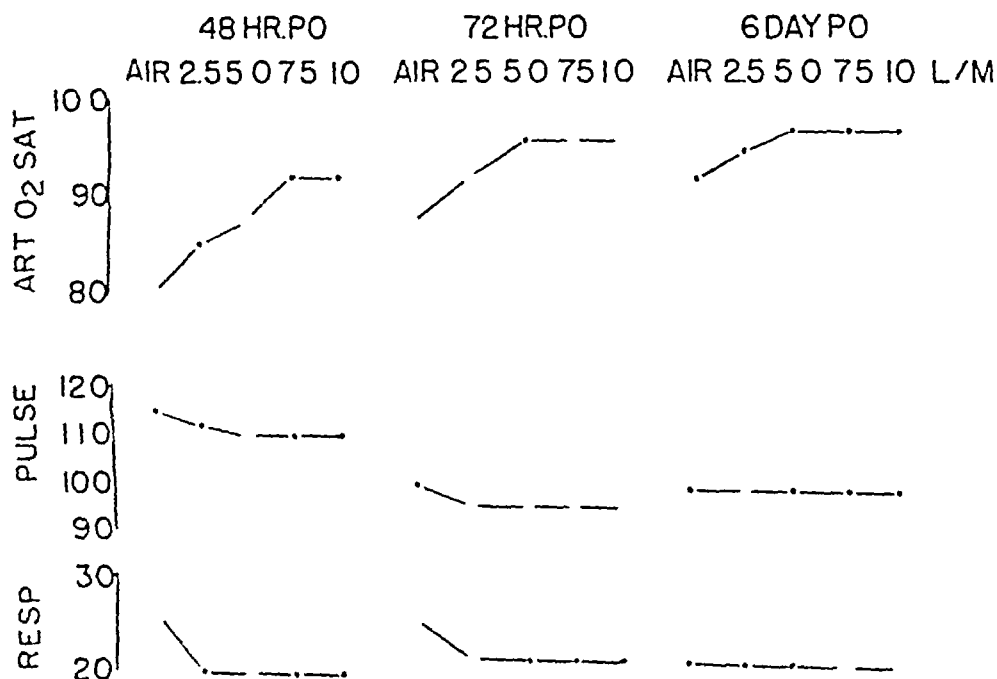
Fig. 2—Response to oxygen via a nasal catheter in a 62 year old patient twenty four hours after a right pneumonectomy. Near to maximum rise in arterial oxygen saturation is obtained with only 2.5 L per minute of oxygen flow. Notice the changes in pulse and respirations closely following the changes in arterial oxygen saturation.

This hypoxia responded quickly to oxygen therapy regardless of the way it was administered. When given 100 per cent oxygen with a McKesson positive pressure machine, arterial oxygen saturation above normal values was reached within one or two minutes after starting. In twenty patients the average increase of arterial oxygen saturation was 10 per cent, a plateau maintained at 98 to 99 per cent levels. Completely similar results were seen when using B L B masks with oxygen flows of 10 to 12 L per minute.

The results in an average case are shown in Fig. 2. It will be noted that oxygen flows of 2.5 L per minute when administered via a nasal catheter proved effective in raising the saturation to normal or close to normal levels in all patients without cardiorespiratory complications, whereas without this information, flows of 5 to 10 L per minute are frequently used. The average increase in saturation obtained in all 50 patients, 24 hours after surgery, was 5 per cent i.e., from 88 to 93 per cent. Five liters of oxygen flow per minute brought a rise in saturation of 7 per cent, from 88 to 95 per cent, or only 2 per cent higher than

that reached using 2.5 L per minute. Higher oxygen flows of 7.5 and 10 L per minute did not prove effective in raising saturation appreciably over the previous levels reached with 5 L per minute in most patients.

JC 567879 RT PNEU + THOR



On admission 27/8/58
 The patient had a right pneumothorax and a right thoracoplasty. Improved
 arterial oxygen saturation with clinical improvement of the patient. Note: poor
 correlation between arterial oxygen saturation and clinical improvement.

In some patients the use of only 1 L per minute oxygen flow via nasal catheter raised the arterial oxygen saturation 10 to 15 per cent, demonstrating how important even small amounts of oxygen are. This finding was only in patients with marked decrease in pulmonary reserve. This was observed preoperatively in patients with advanced pulmonary emphysema. It was seen following pneumonectomy in cases with severe complications such as atelectasis and bronchopleural fistula. Patients with pulmonary hypertension during the postoperative period also evidenced such response to small oxygen flows (Fig 4).

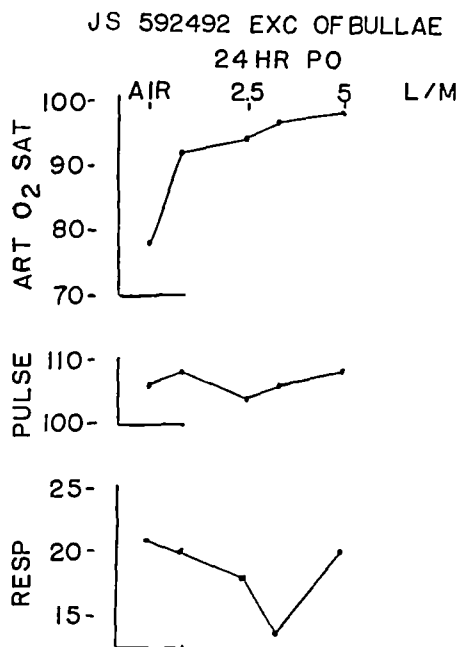


Fig 4—Response to oxygen therapy in a 59 year-old patient 24 hours after resection of left lower lobe for emphysematous bullae. Arterial oxygen saturation raised 14 per cent on 1 L per minute of oxygen flow via a nasal catheter. Notice absolute lack of correlation in pulse respirations and arterial oxygen saturation values.

SUMMARY AND CONCLUSIONS

The use of oxygen therapy as routine procedure is indicated in all post operative cases following major thoracic operations. In young patients with out cardiorespiratory complications, these studies show that there is likely to be no need for oxygen following the day of surgery. In older patients and in the presence of cardiorespiratory complications, oxygen therapy may be necessary for a long period—until the patient is able to maintain an adequate arterial oxygen saturation while breathing room air.

Since adequate arterial oxygen saturation can be maintained on low oxygen flows of from 10 to 50 L per minute, the use of larger amounts of oxygen is not only unnecessary and uncomfortable to the patient, but also dangerous to the extent that it thickens secretions which may lead to atelectasis.

CLOSURE OF EXPERIMENTAL ATRIAL SEPTAL DEFECTS IN SHEEP WITH A SPECIAL ATRIAL RING CLAMP

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INTRODUCTION

A CLAMP has been developed which has proved effective in aiding surgical closure of atrial septal defects in sheep and which promises to prove applicable to human beings¹

The existing surgical techniques, which are amply summarized by Gross and Bjork and Crafoord,³ have fallen into two groups, (a) the extracardiac approach and (b) the intracardiac approach

Because of the ease and safety with which one is now able to palpate the interior of either atrium, it is advisable to use a combination of these two methods. To me, preliminary intracardiac palpation is an essential step in that it at once reveals the anatomical nature of the septal defect present including its site, size, and shape, and all the possible variations of these as fully described by Patten⁴ in 1938. This important aspect having been determined, the surgical closure can then be accomplished.

THE EXPERIMENTAL WORK

On Nov. 6, 1952, experiments were started to find a simple method of closing atrial septal defects.

Sheep, especially lambs and hoggets, were chosen in preference to dogs as being essentially commercial animals with no sentimental value, readily available on the weekly stock market, inexpensive to keep, quiet, and easy to convalesce at Invermay Research Farm, eight miles in the country.

Anesthesia—A half-hour before operation each animal received a subcutaneous injection of morphine $\frac{1}{4}$ grain and atropine $\frac{1}{50}$ grain. Anesthesia was induced with veterinary Nembutal using a dose of 1 grain per 10 pounds of body weight, and injected through a No. 13 B-D needle into one external jugular vein. The needle was left in situ, and through it was threaded a fine polythene tube which was next connected to a glucose saline drip. Muscular relaxation was obtained with intravenous Flaxedil, 1 cc (40 mg). Thereafter, a No. 8 cuffed McGill endotracheal tube was inserted, and the animal connected to an open circuit Palmer pump while an assistant hand operated

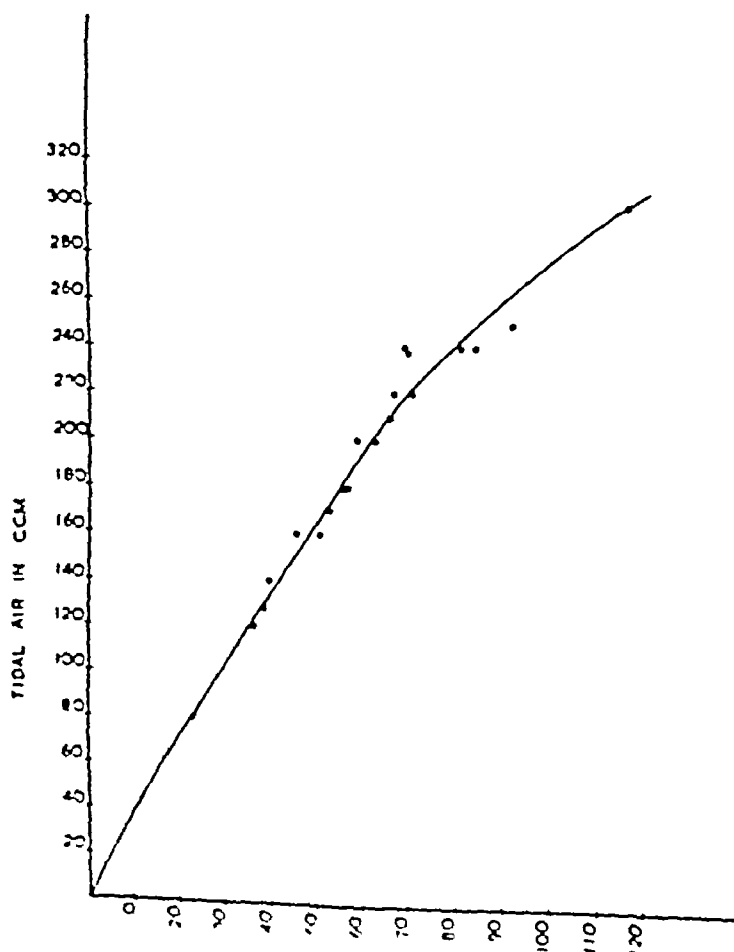
From the Department of Surgery, University of Otago and the Cardio-thoracic Department, Dunedin Hospital, New Zealand.

A 16 mm. color film of this procedure was shown before the Scientific Meeting of the Royal Australian College of Surgeons, Sydney, Aug. 20, 1953.

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the exhaust valve. The closed circuit of the Palmer pump was later used. With the closed circuit the tidal air was adjusted to the weight of the animal as seen in the chart (Fig. 1) which was finally constructed after trial had produced 25 sheep anesthetized with successful recovery.

Phase I—In the first 10 operations the heart was approached by a total splitting of the sternum. The aim was to assess the possibility of approximating the right and left auricular appendages through the normal interatrial septum similar to the method of Swan.



A box-jointed clamp, subsequently found to resemble the clamp that Dodrill⁶ had made to create experimental defects, was fashioned with a central space to accommodate the heart and with two terminal rings of 18 mm internal diameter. Unlike Dodrill's clamp, the rings were not complete circles, each had a gap $\frac{1}{16}$ inch wide in the circumference and placed opposite the attachment of the handle (Fig 2)

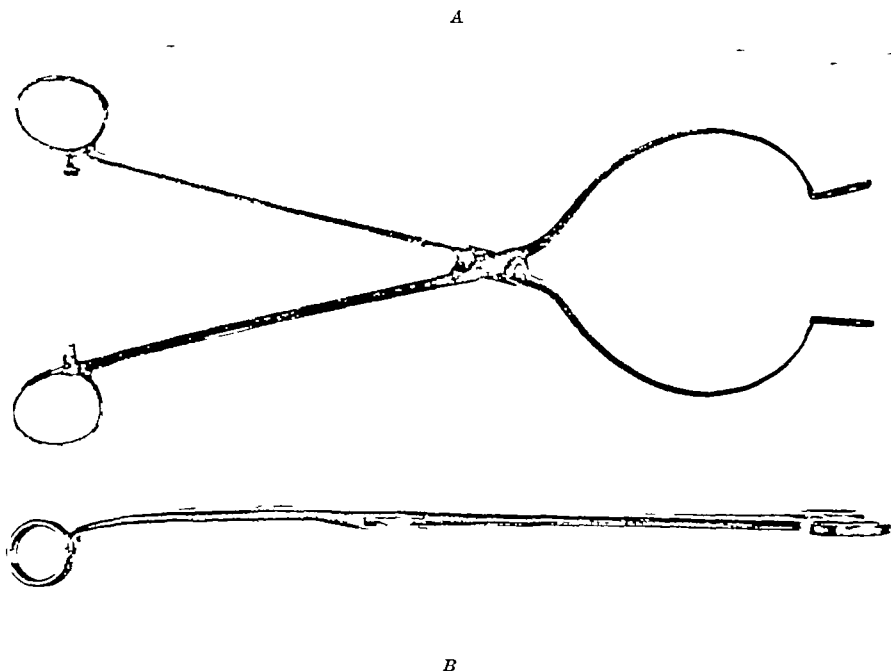


Fig 2—A Atrial clamp from above B side view

The box joint enabled the blades to be removed separately if required, and the gap in the ring allowed safe removal of the clamp in those earlier experiments where one suture had been inadvertently placed over the ring. As only light pressure was needed to close the clamp against intraatrial pressure, accumulating experience showed that the gap in no way weakened the rings. Provided the clamp had not been placed in a position that obstructed the venous return to either right or left atrium, there was no cardiac embarrassment. The surprising feature was how well the heart tolerated the clamp, how regularly it continued to beat and how easily it could be turned from side to side while the sutures were being inserted.

At first one, later two, double thread No 4 silk mattress sutures were passed through the rings from left to right and back again, and tied. Thereafter, the pericardium was closed and a water-seal drain inserted. The sternum was not sutured but the wound was approximated with interrupted silk sutures for the deep layers, and continuous catgut for skin. When the animal had recovered spontaneous respiration, the water-seal drain was removed. Penicillin 200 000 units was given twice daily for five days.

Results of Phase I Seven of the ten animals survived this form of operation. Three died from venous obstruction due to bunching of the auricles from postoperative pleural effusion and from atrial hemorrhage due to misplaced suture after removal of the clamp and at a time when the gaps in the rings had not been made.

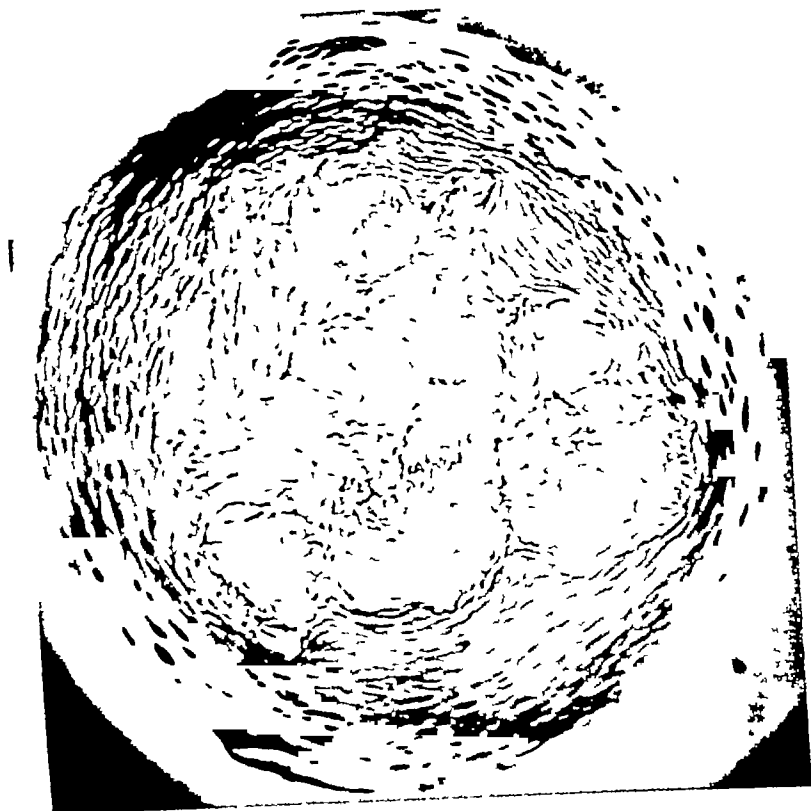


FIG. 1. CLAMP FOR CLOSURE OF ATRIAL SEPTAL DEFECTS IN SHEEP

Comment on Phase I This series of experiments had nevertheless revealed (1) that the atria are as tolerant of being clamped as are the auricular appendages, (2) that, anatomically, there was a considerable space to maneuver in the transverse sinus of the heart which lies in front of the atria and behind the origin of the aorta and pulmonary artery, and (3) that the interatrial septal line lies directly behind the origin of the arch of the aorta in front and to the right of the entrance of the right pulmonary vein behind

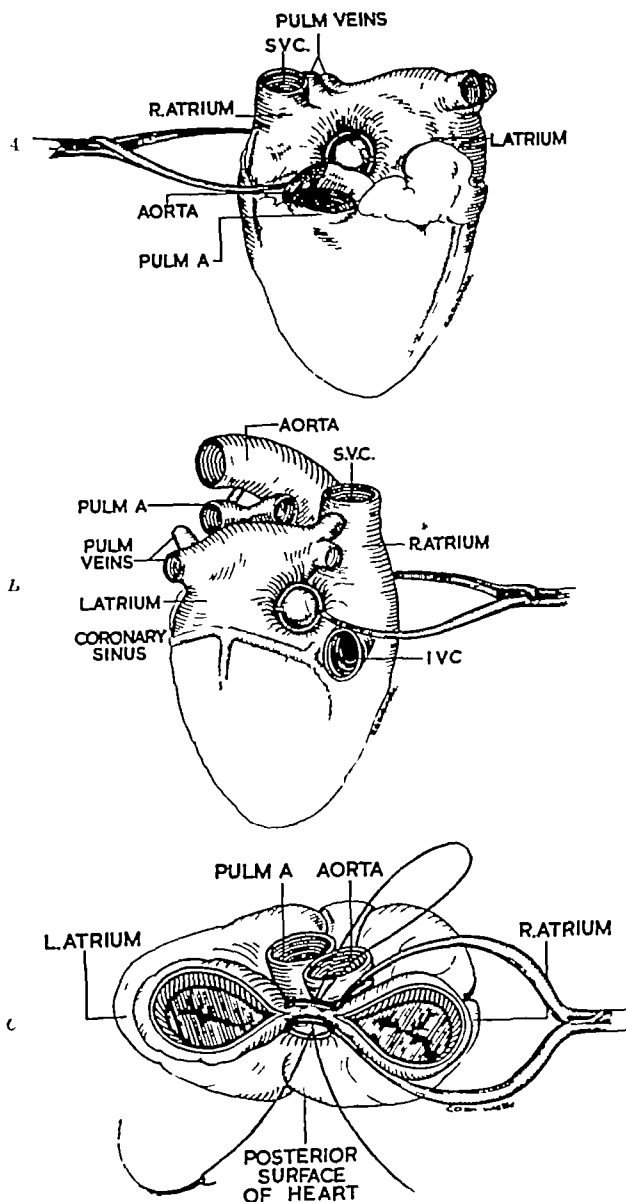


FIG 4 —A The anterior blade in position B The posterior blade in position C Plan from above with one suture passed

Phase II—Realizing these facts, therefore, a second series of experiments was commenced on May 12, 1953. The heart was approached from the right side through the fourth rib bed. One blade of the clamp was placed in the transverse sinus and the other lay behind the heart so as to be *medial* to the line of the superior and inferior vena cavae below the right pulmonary veins and above the coronary sinus. The right pulmonary vein, which was a guide to the *posterior* attachment of the interatrial septum, was defined by dissection. Two or three mattress doubled No. 4 silk sutures were passed from behind forward behind agram, and tied. (Fig. 4.)



two from auricular hemorrhage following dissection of pericardial adhesions, the result of previous operations to create atrial septal defects. Examination of the seven hearts when the animals had been killed two months after operation showed that those sutures placed along the septal line had held, and in that area there had developed a broad sheet of fibrous tissue (Fig 5)

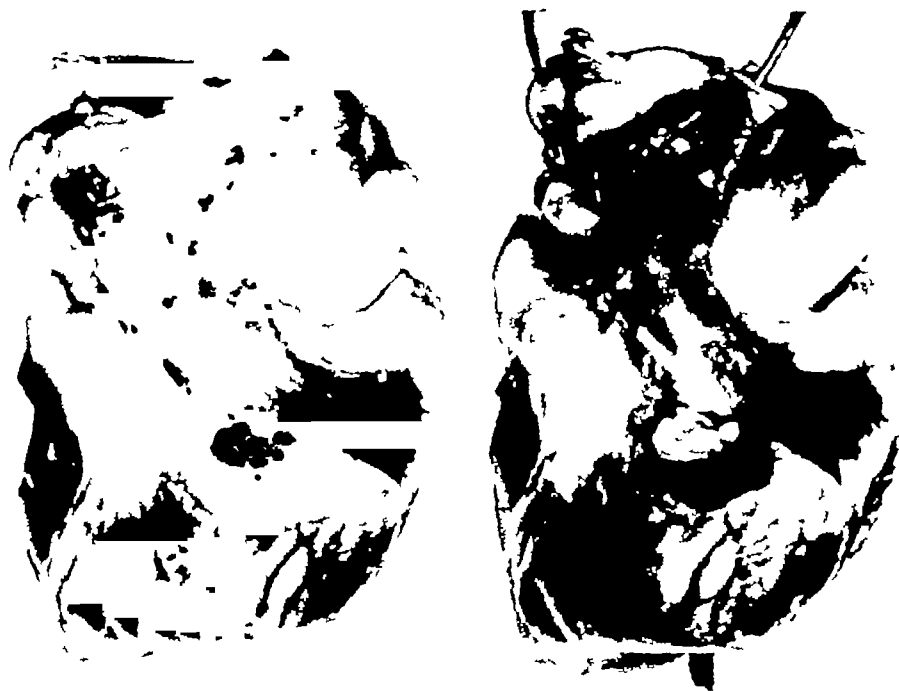


Fig 5—View of interatrial wall from above two months after sutures had been passed

Phase III—To test the method further and give finality to the experiments, it was decided to create atrial septal defects, close them immediately, and thereafter examine the heart, each operation being conducted as an acute experiment. One could thereby assess the degree of closure, undo the sutures, and then see the size of the defect thus closed. The occurrence of pericardial adhesions around all the operated hearts examined two months or more after operation, and the knowledge that experimental defects in time tend to close, supported this attitude.

In four other sheep, the right side of the heart was again exposed, the right auricular appendix clamped, incised, and the right index finger inserted into its lumen. With little pressure it was possible to rupture the septum primum, then remove the index finger, and close the atrial wall with two layers of continuous No. 4 silk sutures. Thereafter the atrial clamp was applied as above, and three mattress sutures were passed from behind forward and back.

again. The sutures were tied from behind and the clamp was removed. The operation was then completed. Immediate autopsy showed that not only were these artificial foramina effectively closed but also when the sutures were untied the foramina had measured on the average 1.5 cm. in diameter (Fig. 6).



It is surmised that this relatively simple method of closing atrial septal defects is applicable to man and that before closing them intracardiac palpation of the defect is advisable to assess the problem

I wish to acknowledge great assistance from Dr F N Fastier and the staff of the Department of Pharmacology, Dr John Ritchie, head of the Department of Anaesthesia, Mr Hall and staff of the animal department, and Miss Marshall of the Department of Photography—all of the University of Otago, and also from Mr George Holmes, Director, Invermay Research Farm, North Taieri, Dunedin, and Mr Moore of the engineering firm, Wilson and Wilson, Ltd, Dunedin

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Recent reports by Holden and Crone⁶ and Cummins and Silver⁷ describe the bronchographic experience with only Dionosil in a total of 128 cases. They presented considerable evidence that only Dionosil is a safe contrast substance for bronchography. No significant complication was encountered among these cases. There was no instance of iodism, either in the case material or after experimental ingestion by animals and human beings. Bronchoscopy following Dionosil bronchography showed no irritative effects on the trachea or bronchi, although one instance of mild clinical tracheobronchitis was mentioned. Coughing during bronchography was no more of a problem than with Lipiodol. Ten of the 128 patients were operated on soon after bronchography, and on pathologic examination of the specimens, no inflammatory reaction attributable to the Dionosil could be found. The postoperative course among these 10 patients was uneventful in all but one. This patient developed right middle lobe collapse which subsequently disappeared. The relation of the collapse to the contrast medium could not be ascertained.

MATERIAL AND TECHNIQUE

The present study is based on a review of the first 30 bronchograms performed with only Dionosil* at the Cincinnati General, Daniel Drake Memorial, and Jewish Hospitals. These were compared as to technical quality, clearance time of the contrast medium, and patient reaction, with an equal number of unselected Lipiodol bronchograms.

The bronchographic technique in the two series was uniform. Patients were premedicated with Demerol, Nembutal, and atropine according to weight. When considerable bronchial secretion was present, postural drainage or bronchoscopic aspiration was carried out beforehand. Local anesthesia was performed by painting or spraying the pharynx with Pontocaine, and injecting 2 to 5 cc of 10 per cent cocaine into the trachea through a cannula. General ether anesthesia was used in children under 10 years of age.

When satisfactory anesthesia was obtained, a tracheal catheter was inserted to the region of the carina. The medium was injected at room temperature, the Dionosil requiring thorough shaking before use. In the average adult approximately 15 cc of contrast medium was instilled into each lung. Proportionately smaller amounts were used in children. In patients with poor respiratory reserve and in those in whom filling of only one lung was indicated, unilateral bronchography was performed. In all others complete mapping of both lungs was attempted at one sitting. During the injection the patients were postured in the usual manner, and the adequacy of filling was checked fluoroscopically. Usually several spot films were taken at this time. When filling of one lung was complete, recumbent anteroposterior and lateral Buck films of the chest were obtained. The opposite lung was then filled and examined in a similar manner, but right and left posterior oblique views were substituted for the lateral. At the completion of the procedure, postural drainage was instituted.

*Supplied through the courtesy of Picker X-Ray Corporation, White Plains, N. Y.



Fig 1—Lateral bronchogram of right lung (selective bronchography). Collapse of middle lobe and anterior and medial segments of lower lobe in slowly resolving pneumonia. Collapsed segments are well filled with Dionosil



Fig 2—Normal Dionosil bronchogram in a child (lung mapping). All segmental bronchi were visualized

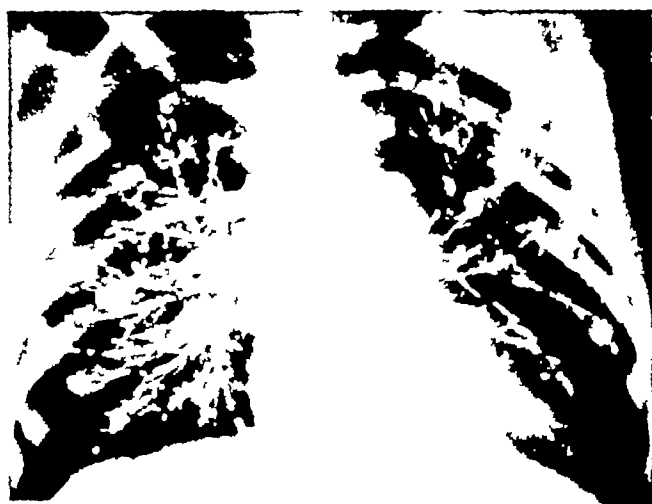


Fig. 5. Distribution of oily dionosil in bronchile cysts. The anterior basal and lateral basal segments in the left are incompletely filled.



Fig. 6. Distribution of oily dionosil in bronchile cysts. The anterior basal and lateral basal segments in the left are incompletely filled.

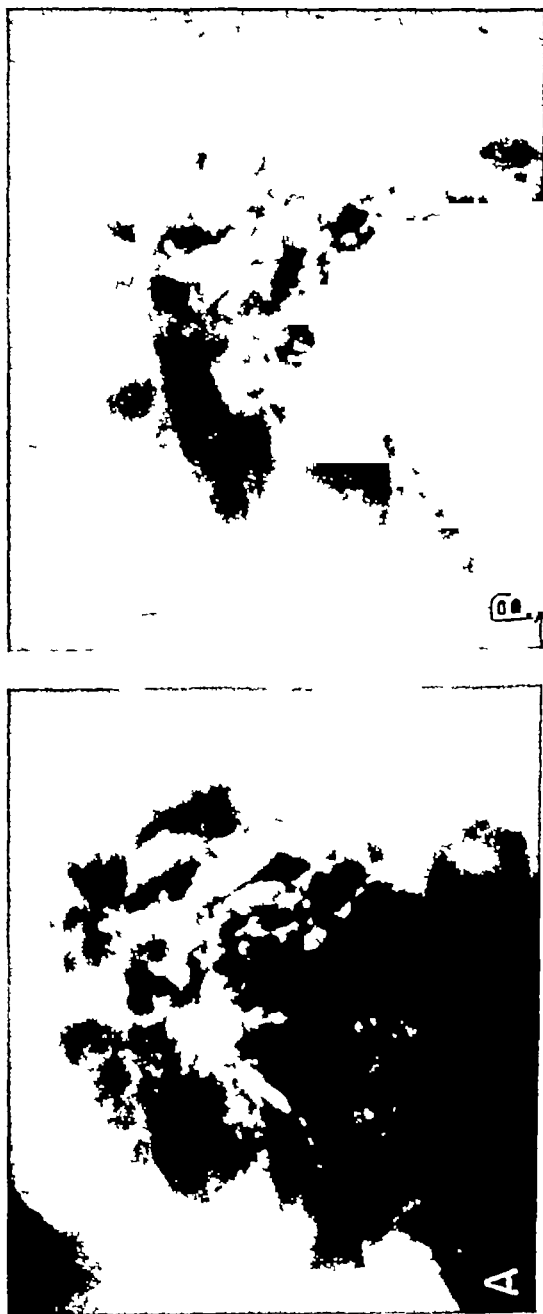


Fig. 5 —Bronchiectasis right upper lobe A Dionosil B 1 pipelot

Holden and Crone¹ stated that alveolar filling did not occur with oily Dionosil. We have observed alveolar flooding with both media although the Lipiodol did show a greater tendency to do so. There were 18 instances of flooding with Lipiodol compared to 7 with Dionosil (Fig. 4).

The relative ability of the two contrast media to coat the bronchial wall was compared. It was found that Dionosil more commonly produced a double contrast effect (Fig. 5, P₁). However, it was also noted that mucosal coating occurred more frequently when smaller amounts of either contrast substance were present in a bronchus, and this was especially true on films made near the end of the examination. A fairly widespread double contrast effect was noted in 15 Dionosil but in only 2 Lipiodol bronchograms. We do not, however, consider bronchial coating an important advantage.



The most important advantage of Dionosil over Lipiodol is the rapidity with which it disappears from the chest roentgenogram. Films were made approximately 24 hours after Dionosil bronchography in 29 instances, and after 48 hours or more in 8. The average amount of clearing of Dionosil in 24 hours was about 90 per cent. Complete disappearance occurred in 48 to 72 hours (Fig 6). The slowest clearance rate encountered was 70 per cent in 24 hours.

In 18 Lipiodol cases, follow-up films were available varying from 1 day to 19 months after bronchography. The Lipiodol often remained in the lungs for weeks and even months. Retention was particularly striking in amount and duration in areas of emphysema and in segments where flooding had taken place (Fig 6). The persistent shadow of the iodized oil often masked a parenchymal lesion, making serial film comparison difficult or impossible. It was particularly troublesome in the interpretation of postoperative roentgenograms and repeat bronchograms.

In seven Dionosil cases material for histologic study was available 1, 5, 5, 6, 7, 8, and 51 days, respectively, after bronchography. In addition to routine paraffin sections, fat stains were prepared in six cases. In three of these (two with carcinoma and one with bronchiectasis) chronic inflammatory changes were present. Intracellular fat droplets were visible, but it was impossible to determine whether this fat indicated endogenous lipoid pneumonia, a condition found in such cases, or represented phagocytized peanut oil. In two of the three (1 and 51 days, respectively, after bronchography) free oil, believed to represent residual contrast medium, was seen in the alveoli and bronchi. The remaining three specimens, obtained 5, 7, and 8 days, respectively, after bronchography, revealed free fat droplets in the bronchi and alveoli with no surrounding inflammatory reaction. There was no intracellular oil.

Although our pathologic material is limited, it appears that only Dionosil causes no significant inflammatory reaction in the lungs. Cummins and Silver⁷ stated that there was less oil in the lungs after Dionosil than after Lipiodol bronchography, and attributed this to the fact that Dionosil was less viscous and, therefore, more readily expectorated.

DISCUSSION

Only Dionosil compares favorably with other contrast media used for bronchography. Like Lipiodol, it is much less irritating to the tracheobronchial tree than the water-soluble media, judging from the reports on the latter in the literature. Good anesthesia, therefore, is readily obtained, and cough presents no serious problem.

There is no significant difference between Dionosil and Lipiodol in contrast, density, or detail as seen on the roentgenogram (Figs 5, 7, and 8), and both are superior in these qualities to the water soluble media. However, Dionosil and the water soluble media are slightly more difficult to see fluoroscopically than Lipiodol.



Fig. 1. Case 1 (Fig. 10 of Hilson et al.). Oblique Dionosil bronchogram reveals deformity and obstruction (central and ant. for. segmental branch).



A

B

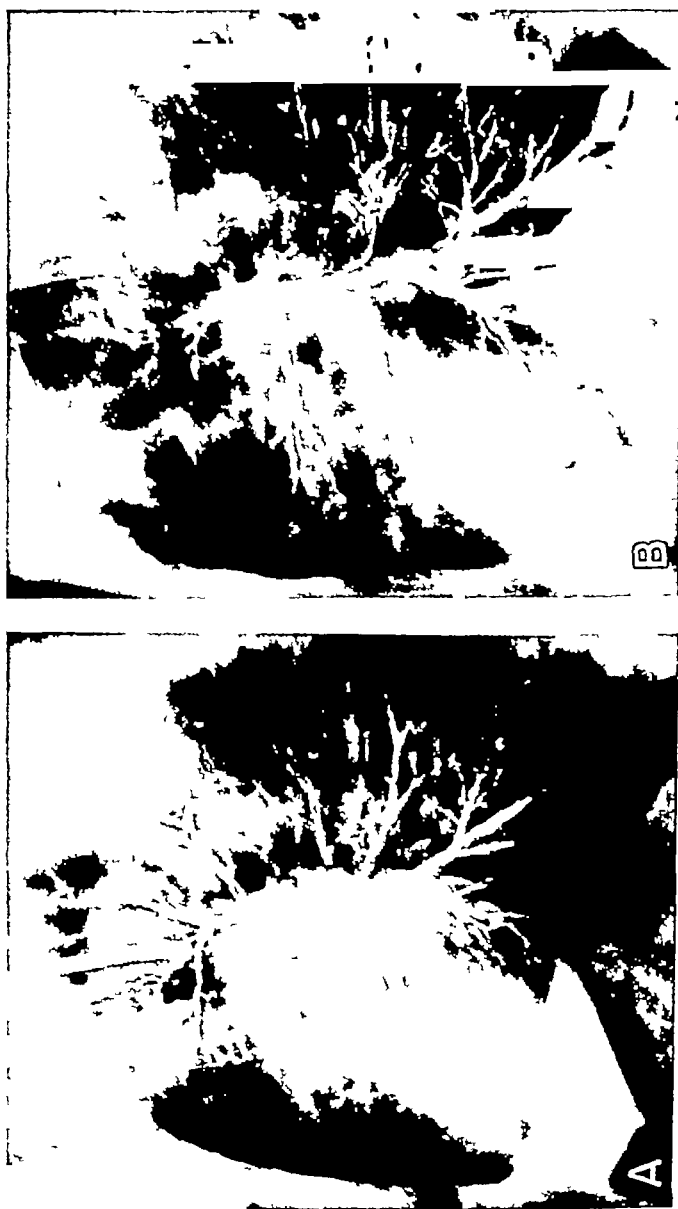


Fig. 9—Internal Bronchoscopy. A Incomplete visualization of peripheral bronchi. B Delayed film one hour later showing improved peripheral filling.

Dionosil presents the major advantage over Lipiodol of rapid disappearance from the roentgenogram. It clears somewhat more slowly than the water-soluble media. This is also an advantage since it is not necessary, as it is with water-soluble media, to hurry the examination in order to avoid a blurring and smudging effect which obscures the detailed delineation of the bronchi. However, moderate delay after Dionosil instillation may also cause this effect. It only occurs when repeat or delayed films become necessary for technical reasons. Nevertheless, we obtained an excellent delayed bronchogram without smudging one hour after Dionosil instillation in one of two attempts (Fig. 9). The rapid clearing of Dionosil virtually complete in 24 hours is in part due to its low viscosity which more readily permits its expectoration and to the speedy absorption and excretion of the iodide.

There has been no evidence of harmful effects from Dionosil although the number of cases studied is too small to conclude that the medium is entirely innocuous. To date neither pneumonia, pulmonary granuloma, nor iodism have occurred. The fate of the peanut oil in the lungs requires further study, but considerable experience with Iodochlorol, an iodized and chlorinated peanut oil as a bronchographic medium indicates that this oil is relatively harmless. Fouch, Basil, and Davis, utilizing radioactive tracer techniques, found no free inorganic iodide in the urine after Dionosil bronchography. The contrast medium is excreted entirely as an organic iodide. Iodism has been attributed to the presence of the free iodide ion^{11, 12} and therefore should probably not occur with Dionosil. The combined experience with resected lung specimens after bronchography with only Dionosil which amounts to only 17 cases including the seven in the present series, revealed no apparent gross or microscopic damage to the lung.

The optimal time interval between bronchography and operation has not been established for Dionosil, but judging from present indications it could appear that a waiting period is not necessary.

Only Dionosil has all the essential attributes of Lipiodol and water-soluble media without their disadvantages. We feel that this medium or one similar to it will ultimately replace those currently used for bronchography.

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Multiple microscopic sections were prepared from each area of injection. There were 5 specimens for each of the 4 dosages and 7 different intervals, so that in all 140 different sites of injection were studied. In general, the severity of the reaction varied, but not directly so, with the amount of Efocaine injected. The changes noted were as follows:

Five minutes There were no changes noted in any of the 20 nerves examined.

Twenty-Four Hours The majority of the nerve sections did not reveal any changes, but a few showed swelling of the myelin sheaths, and two showed minor hemorrhage.

Seventy-Two Hours General nerve structure was still normal, but vacuolization, swelling and basophilic staining were present.

Seven Days All nerves showed severe destruction as evidenced by focal hemorrhage, vacuolization, partial absence of fibers, nuclear fragmentation, and disruption of nerve sheaths.

Fourteen Days Same changes were present as for seven days except that in a few sections perineurial fibrosis was beginning to occur.

Twenty-One Days Severe destruction was still present as evidenced by complete lack of organization of the nerves. Nerve fibers were not identifiable as such, much vacuolization was present, some few capillaries were noted in the nerve bundles (Fig. 1).

Twenty-Eight Days Nerves were recognizable, although edema was still present. Many sections showed small nerve tract formation and myelin axon regeneration.

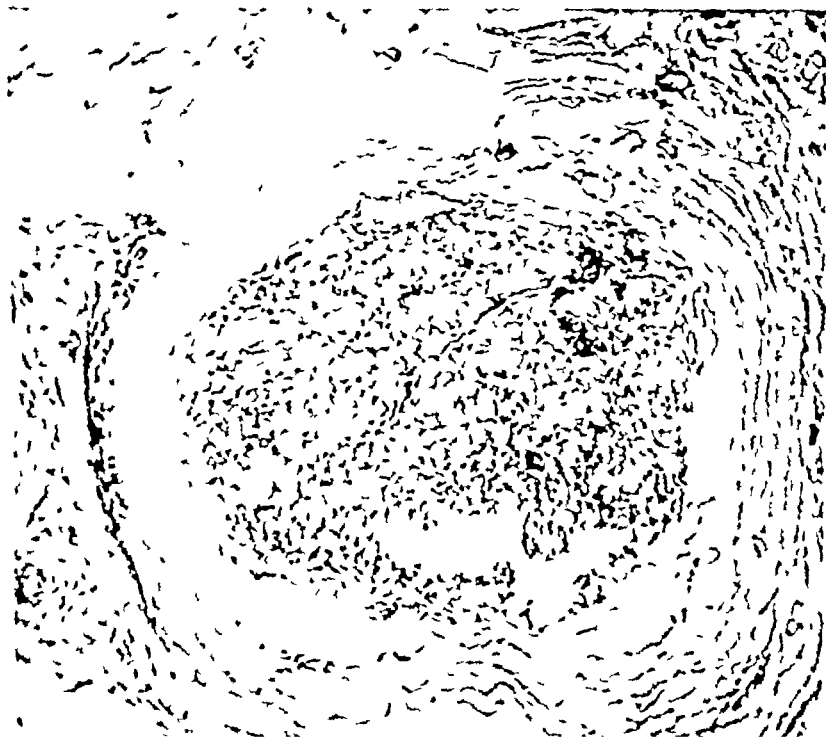
The effect of Efocaine on muscle, artery, vein, subcutaneous tissue, fat, pleura, and periosteum was also noted. The muscle tissues were usually fragmented, hemorrhagic, and infiltrated with phagocytes. The reaction was always more severe in the muscle than in the nerve. The arteries and veins frequently showed endothelial proliferation and/or thrombosis, particularly when the larger doses were used. The other contiguous tissues showed little or no reaction.

COMMENTS

It was immediately apparent that the anatomical changes produced by Efocaine were more pronounced than the physiologic changes. At 21 days, for example, even though the nerves appeared to be completely disrupted, each one had regained its function as judged by ability to transmit both afferent and efferent impulses. Nevertheless, the reaction produced is severe, and prolonged neuritis theoretically might well occur following such a severe chemical reaction.

Paraplegia did not occur in any animal receiving an intercostal nerve block with Efocaine. Yet it has been reported several times as following intercostal block in human beings. Explanations so far for the development of such paraplegia have been based on the assumption either (1) that Efocaine reaches the spinal cord by capillary action along the nerve fibers themselves or (2) that Efocaine was actually injected into a perineurial dural extension. While the former may have some basis, as shown experimentally by Moore, the latter does not, for introduction of Efocaine into the subarachnoid space itself produces

only a prolonged spinal anesthesia (10 to 14 days). Batson's work¹⁴ which demonstrated the collateral circulation between the intercostal and spinal veins suggested an inquiry into the possible effect of the introduction of EPOCAINE into the intercostal vessels. It is easy to understand how intravascular injection may occur during initiation of an intercostal nerve block with the vessels being in such close proximity to the nerve. Also it is well known that lack of aspiration of blood through a needle does not guarantee that the tip of the needle is not in a vessel. As a preliminary experiment 3 cc. of EPOCAINE were introduced into an anesthetized dog's exposed right fifth intercostal artery and into another



RESULTS

It was noted that successful injection into either the intercostal vein or artery was accompanied by extreme fasciculation of the muscles on the injected side below the level of injection. Thus, it was possible to know that all injections were successful. Fourteen dogs were injected, seven in the artery, seven in the vein. At intervals varying from 10 minutes (time required to expose the cord after the injection) to 5 weeks, the spinal cord was removed and microscopic sections made of the involved area.

Paralysis was present to some degree in 12 of the 14 dogs so injected. In some cases both hind legs were partially affected, in others paraplegia was present. In two dogs only the limb on the injected side was involved, two other dogs did not show any involvement. The duration and severity of the paralysis varied, but in every animal that was to show improvement, it became evident by the third day.

A brief summary of the lesions present in each dog is presented in Table I.

TABLE I. SUMMARY OF EXPERIMENTS—INTRAVASCULAR INJECTION OF EPOCAINE

I Intercostal Arterial Injections

Dog No 1513 was sacrificed immediately while still anesthetized. There was no cord, vascular, or meningeal lesion.

Dog No 1517, sacrificed at 24 hours, had paralysis of the left hind leg, edema of central area of cord, but no vascular or meningeal reaction.

Dog No 1518, sacrificed at 24 hours, was paraplegic, had necrosis and hemorrhage of almost the entire cross section of the cord, necrotizing and exudative thromboangitis, and the meninges showed acute inflammatory changes.

Dog No 1512, sacrificed at 4 days, was paraplegic, there were necrosis and hemorrhage in the left anterolateral section of the cord. The vessels showed a necrotizing thromboangitis, and the meninges presented acute inflammation and proliferation (Fig 2).

Dog No 1509, sacrificed at 7 days, had bilateral partial paralysis, with it being more marked on the left. Microscopic sections showed cystic necrosis of the posterior half of the cord, exudative and proliferative angitis of the vessels, and chronic inflammation of the meninges.

Dog No 1515, sacrificed at 7 days, also had bilateral partial paralysis, more marked on the left. Microscopic sections revealed necrosis and hemorrhage of the posterior central part of the cord, slight exudative angitis of the vessels, and no meningeal reaction.

Dog No 1502, sacrificed at 35 days, had bilateral partial paralysis, with more involvement on the left. Microscopic sections revealed cystic necrosis of the central part of the cord, proliferative arteritis of the vessels, and hemosiderosis and chronic inflammatory changes in the meninges.

II Intravenous Injections

Dog No 1514 was sacrificed without recovery from anesthetic. Microscopic sections did not reveal any lesions of the cord, vessels, or meninges.

Dog No 1510, sacrificed at 24 hours, was paraplegic. Microscopic sections revealed necrosis and hemorrhage of the left half of the cord, necrotizing thromboangitis of the vessels, and acute inflammation of the meninges.

Dog No 1511, sacrificed at 24 hours, was paraplegic. Microscopic sections presented necrosis and hemorrhage of practically the entire cross section of the cord, necrotizing thromboangitis of the vessels, and hemorrhage and inflammatory changes in the meninges.

Dog No 1508, sacrificed at 4 days, was paraplegic. Microscopic sections revealed necrosis and hemorrhage of the anterior central portion of the cord, necrotizing thromboangitis of the vessels, and hemorrhage with moderately acute inflammatory changes in the meninges.

Dogs Nos 1519 and 1520, sacrificed at 7 days, had no signs nor was any lesion of the cords found at autopsy. (Each dog presented signs of a successful injection at the time of injection, but was never paralyzed.)

Dog No 1501, sacrificed at 35 days, had bilateral partial paralysis, more marked on the left. Sections revealed cystic necrosis of the right posterocentral portion of the cord, with no involvement of the vessels or meninges (Fig 3).



SUMMARY

Efocaine, when used to induce an intercostal block in the dog, produces necrosis of the nerve and the contiguous tissues. This reaction is more severe anatomically than physiologically. The injection of Efocaine into an intercostal vessel produces myelitis of a varying unforecastable degree. Whether this effect is produced by chemical necrosis of the cord or by chemical thrombosis of the cord blood vessels was not determined.

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EXPERIMENTAL FUNCTIONAL SEPARATION OF DOG LUNGS

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New York It essentially consists of two thin-walled brass tubes soldered together with distal ends flared so that these ends just enter the main bronchi beyond the carina. A smaller metal tube soldered into the groove between the two main tubes conducts air pressure to a carefully molded balloon which encircles the distal end of the apparatus. This balloon is designed to press tightly against the tracheal walls as well as the carina, thus separating the gas flow of the lungs. The divider is maintained in position by rubber bands stretched between the proximal side vent tubes and the animal's canine teeth to force the tubes and balloon continually caudad to ensure a tight seal. The entire divider is therefore passed through the larynx and impinges upon the carina, obviating tracheotomy.

Fig 1, *a* shows this divider in place in the scale drawing of the average dog. It may be seen that the right upper lobe bronchus lies just beyond the balloon, even proximal to the metal tip, so that it may become obstructed. This and slipping of the tight carinal seal necessitate careful adjustment of position of the divider initially as well as proper maintenance of position during an experimental procedure. It is important to obtain proper oxygen consumption values for each lung prior to and after any experiment which assumes the divider is properly in place.

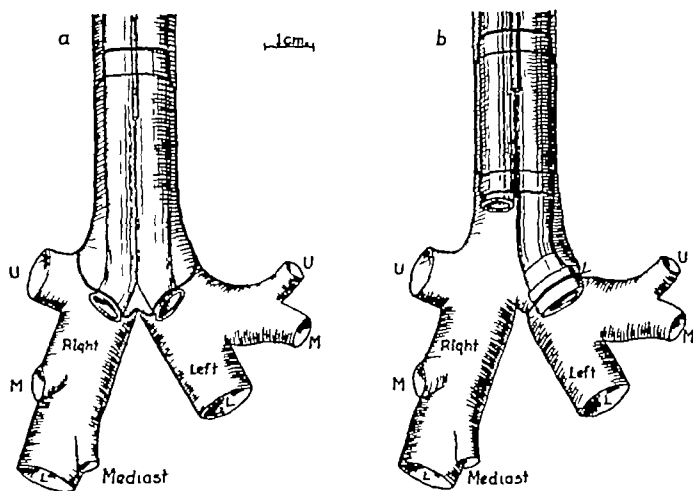


Fig 1—Scale drawing of trachea and bronchi from fourteen separate measurements in each of twelve dogs. In *a* the Wright divider is in place while in *b* the bilumen plastic catheter is tied in. Upper middle and lower lobe bronchi are labeled U, M, and L respectively. In addition there is the mediastinal lobe bronchus on the right side.

Further disadvantages include difficulty in making and properly fitting the balloon, frequent balloon leaks, and the necessity to replace the balloon after a few experiments. With experience however it was possible to place the divider rapidly and to maintain separation of the lungs so long as there was no large pressure difference between the lungs. In certain experiments^{1, 2} the Wright tracheal divider has been very satisfactory and is still used routinely in this laboratory for certain types of experiments. It has the ad-

average. Let animals are not harmed by the procedure and can be used as often as necessary. Analysis of the relative oxygen consumption and ventilation of the two lungs using this device indicated close correlation with the relations of the weights of the two lungs, namely, 60 per cent for the right and 40 per cent for the left lung.

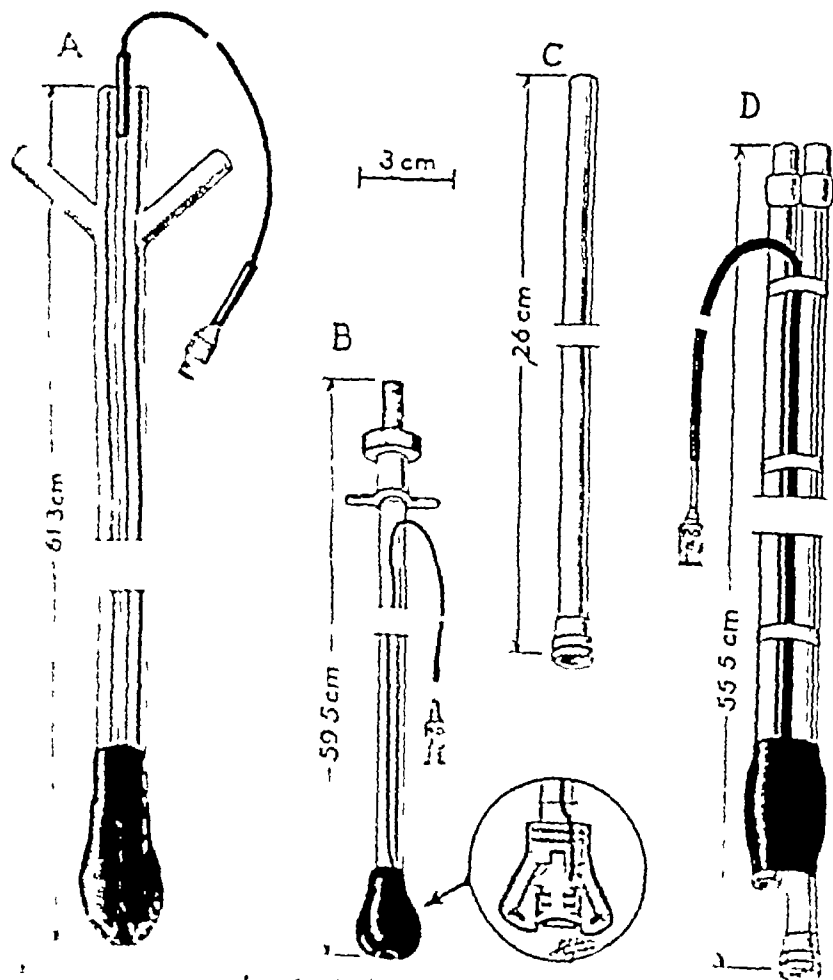


Fig. 1. Diagram illustrating the functional separation of dog lungs using a device. A, B, C, and D are the components of the device. A, B, and C are the tubes. D is the Y-shaped connector. The dimensions are in centimeters.

be done in a lobar bronchus. Addition of an inflatable balloon did not serve to anchor the device properly in the main bronchus, and with continued failure at this site, attempts were ended.

3 *Transthoracically Placed Endobronchial Cannula*—A small series of animals were prepared by tying a large lumen plastic cannula (Fig 2, c) within the left main bronchus after severing it from the trachea within the chest and closing the tracheal defect. With care the trachea could be made airtight. The cannula was then led out the chest wall, in various positions, both anterior to and posterior to the aorta or its arch. Invariably the cannula tip impinged against the left pulmonary artery, either entirely or partially blocking its blood flow (determined either by inspection or by oxygen consumption study). The device was therefore discarded.

4 *Bilumen Plastic Endotracheal Cannula Tied Within Left Main Bronchus*—This method was eventually devised and in our experience surpassed previous ones in most respects. The cannula was easily made of two Tygon plastic tubes whose distal ends were bound together with adhesive tape over a Lucite form lying between. A metal tip protruded from within the end of the longer side (Fig 2, d) with a groove to permit a ligature to lie in place. A balloon about the distal end could be inflated by means of pressure via a small polyethylene tube. Fig 1, b shows this device in place in the scale drawing of the average dog, the longer tube projecting into the left main bronchus where it is tied securely, the shorter tube lying in the trachea and serving the right lung.

This bilumen plastic cannula was placed through a tracheotomy and the balloon inflated to allow intermittent pressure during the ensuing thoracotomy. The animal was then placed left side up and the left thorax entered through a high intercostal incision. After opening the mediastinal pleura anterior to the aorta and posterior to the left vagus nerve, the left main bronchus was bluntly dissected up out of the mediastinum and the tube pushed down into it. A heavy silk suture was tied about the metal tip lying in the left main bronchus to retain the tube exactly as placed. Prior to closure of the chest the left tube was temporarily disconnected from the pressure source to ascertain that the upper lobe bronchus was not obstructed and that the lobe would collapse as pressure was removed. Occasionally the tube was changed to a more proximal position when this occurred. The chest was then closed in layers about a catheter in the pleural space which was used to remove pneumothorax air at conclusion.

After some experience with this method the preparation could be made in an average of thirty minutes, often in less time. The animals survived in good condition under anesthesia until sacrifice as long as eight hours later. There was no instance of mucus plugging of the tubes. Once the tube slipped out of the ligature, and was replaced successfully by reopening the chest. Once in place the animals could be moved or turned as desired with assurance that the lungs were separated not only from each other but also from the outer air. In twelve instances data were obtained for the right and left lung tidal volumes and oxygen consumptions. The averages are indicated in Table

VENTILATION OF THE OPEN LUNG DURING UNILATERAL EXPERIMENTAL ATELECTASIS

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PREVIOUS studies in this laboratory have reported our findings concerning the principles governing gas absorption from the blocked off lung in the early phase of atelectasis,¹ the evolution of more satisfactory methods for separation of dog lungs to allow individual lung function experiments,² and the effects of atelectasis of one lung upon the volume and the pressure volume relationship of the contralateral lung.³

The present study was made to evaluate the causes which precipitate rapid changes in the frequency of breathing and contralateral tidal volume increase whenever unilateral atelectasis is produced experimentally. Similar changes may be observed in human beings with sudden atelectasis (particularly when this is of major degree).

EXPERIMENTAL DESIGN

Directly following a major degree of atelectasis such as that of an entire lung, the pulmonary tissue available for gas exchange is markedly decreased. One would assume that adjustments in ventilation would follow, probably in the direction of increased ventilation of the open lung. Observations of experimental animals indicate that this does occur and can be measured in terms of increased rate of respiration, increased tidal volume, and a larger minute volume of the open lung. In fact the minute volume of the one open lung now becomes larger than the total minute volume of both lungs prior to atelectasis.

This increase in total pulmonary ventilation might be attributed to various mechanisms acting alone or together. First, previous experimental data have indicated an immediate contralateral tidal volume increase following unilateral atelectasis due to the "mechanical effect" of blocking one bronchus.³ The mechanical increase does not, however, explain all the increase in contralateral tidal volume that occurs. It has been apparent that besides this immediate change which follows upon bronchial occlusion, there is a slow increase in tidal volume to approximately twice that of the control level. Thus, all of the tidal increase is not due to the "mechanical effect." Furthermore, the mechanical effect cannot explain the increase in rate of respiration.

Two other factors may be considered, namely reflex changes probably mediated over the vagus nerves and blood chemical changes acting on the

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inflating the balloon the left pulmonary artery could be blocked. Since there was a possibility that the balloon might slip down into a distal branch of the pulmonary artery, a second method was used later, by which the whole artery could definitely be blocked and released. A heavy silk tie was placed loosely about the left main pulmonary artery and tied into the posterior thoracic wall tissue. Several long silk ligatures were then passed about the artery and led out the chest wall. When pulmonary artery occlusion was desired a long ligature was pulled up tight and clamped outside the chest wall. Release was accomplished by cutting and pulling out the suture. Subsequent arterial block was effected by repeating this maneuver with a second long suture and so forth.

Atelectasis was obtained either by blocking the left lung cannula or by rapidly aspirating all gas from the left main bronchus cannula lumen and clamping the end. At conclusion further unsuccessful attempts to remove gas ensued that no leak had occurred.

Standard *metabolism machines* containing CO_2 absorber could be connected either to one or both lumens of the bronchial cannula to allow measurement of O_2 absorption, rate, tidal volumes, and minute volumes.

RESULTS

Fifty-eight experiments were performed on forty animals. The results may be divided into two groups.

1 Complete O_2 consumption data were obtained during the development of atelectasis in nine experiments on seven animals. Initially both lungs were connected to spirometers and the O_2 consumption of each lung measured over a minimum period of ten minutes. The final ten minute period before initiation of atelectasis served as a control. Left lung atelectasis was begun by bronchial occlusion and right lung O_2 consumption determined for a minimum period of thirty minutes. Left lung atelectasis was then reversed simply by removing the clamp from the left main bronchial cannula and allowing the left lung to resume function and to re-aerate. O_2 consumption in both lungs was measured then for a minimum period of twenty minutes. Fig 1 illustrates the averaged findings. During the latter part of the control period the right lung O_2 consumption was about 50 per cent of the total. When left atelectasis was initiated by clamping the left lung cannula, the right O_2 consumption as indicated by the graph rapidly increased about 50 per cent of its control value, then over a six-minute period reached the control total O_2 consumption value and thereafter exceeded this by about 6 per cent.

When the left lung was allowed to re-expand, the right O_2 consumption rapidly at first and more slowly later returned to approximately preatelectasis values. The total O_2 consumption likewise returned to the control value which is indicated by the broken line on the graph.

2 Forty-nine experiments in thirty-three animals were performed to evaluate the vagal reflex and blood chemical control of respiration during unilateral atelectasis. The summary of these data (averaged) is indicated in Fig 4 as five types of experiments. The respiratory rates and tidal volumes

tube The horizontal broken line indicates the total oxygen consumption during the control period The unshaded area represents the oxygen uptake of the left lung before occlusion It is about 50 cc per minute Immediately upon occlusion it is apparently reduced to 25 cc per minute and six minutes later is reduced to zero as indicated by area A

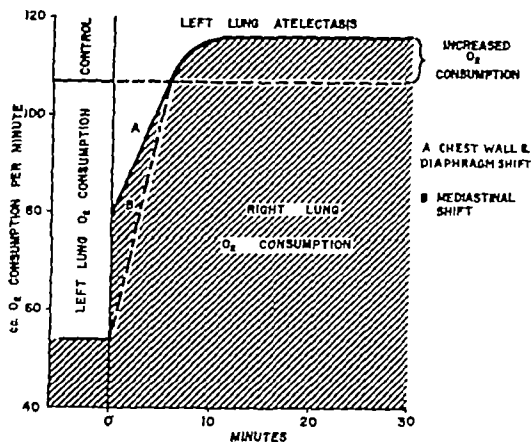


Fig 2—Diagrammatic form of Fig 1 showing O_2 consumption study before during and after left lung occlusion (See text)

Analysis of the events accompanying lung collapse under these conditions, however, provides a different picture and indicates that the spirometer attached to the open right lung is now not only measuring right lung O_2 consumption but also is indicating about 50 per cent of left lung O_2 consumption. It has been shown elsewhere³ that when gas is removed from the left lung by aspiration about 50 per cent of the volume decrease can be accounted for by increase in open right lung volume due to a mediastinal shift. It has been assumed that the remaining 50 per cent volume change is accounted for by inward movement of the chest wall and cephalad movement of the diaphragm. Fig 3 illustrates these shifts diagrammatically.

Therefore the sudden *apparent* increase of 25 cc per minute right lung O_2 consumption in Fig 2 represents not only the *true* right O_2 consumption but also 50 per cent of the left O_2 consumption (since this continues until O_2 is completely absorbed from the left lung). Area B in Fig 2 thus represents overdistention of right lung and mediastinal shift, while area A is the chest wall (including diaphragm) shift, and A and B are approximately equal. The ascending broken line of Fig 2 thus marks the true right lung O_2 consumption. At six minutes after left lung airway occlusion, the right lung carries the total O_2 consumption and the left lung must be collapsed. This is ample time if one assumes that the blood flow to the collapsing lung is maintained. The functional resting volume of our dogs is approximately 150 cc and the left lung circulation normally removed about 50 cc per minute in our control run.

Fig 2 also allows one to compute the total volume of the left lung from areas A and B. Initially the O_2 uptake was 50 cc per minute and was re-

rate and tidal volume of the right lung is unable to compensate completely since the $p\text{CO}_2$ rises as judged by the fall in pH. The increase in over all O_2 uptake would likewise require a 6 per cent increase in alveolar ventilation to account for the greater air exchange for this O_2 consumption.

Chemical Factors—The function of one lung can be eliminated either by acutely cutting off the pulmonary artery blood flow and permitting the ineffective ventilation to continue or by acutely stopping the ventilation (atelectasis) and permitting the ineffective pulmonary blood flow to continue. In either case the contralateral open lung must take over the entire function of gas

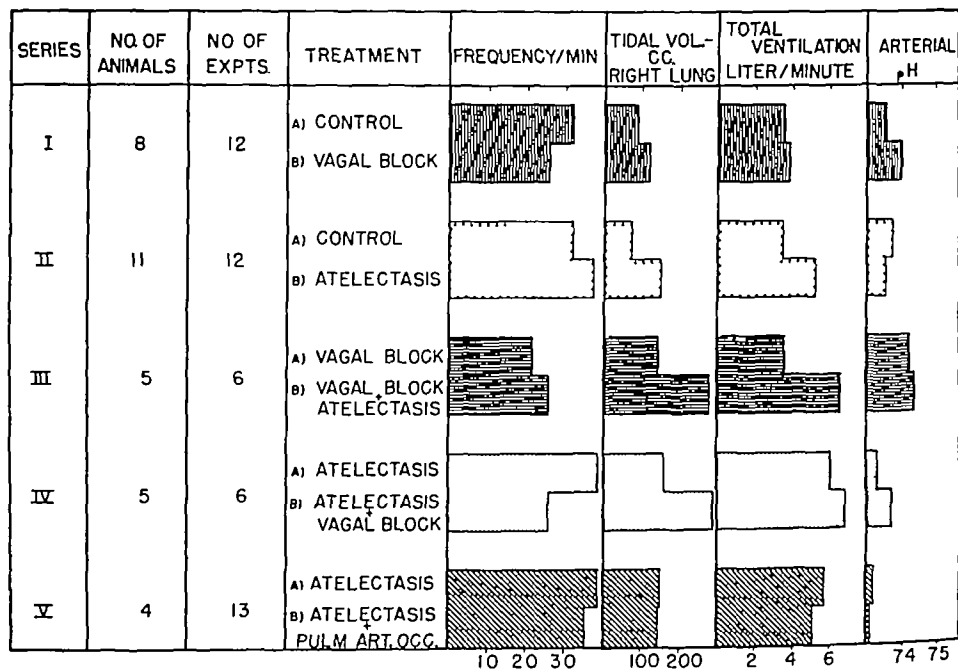


Fig 4—Average data of forty nine experiments on thirty three dogs to assess the differences in open lung ventilation between two sets of conditions as indicated

exchange by approximately doubling the alveolar ventilation if the carbon dioxide tension or pH of the arterial blood is to remain as before. If, however, the blood flow through the atelectatic lung is not stopped completely,⁶ then only the CO_2 level of the arterial blood can be compensated for by hyperventilation while O_2 desaturation cannot. The mixed arterial O_2 level (breathing O_2 or air) must fall in proportion to the magnitude of the shunt since the hyperventilation would not appreciably increase the arterial O_2 content on the contralateral side.

Observations of the ventilation after sudden occlusion of the left pulmonary artery in dogs show that the ventilation is increased and partially compensated⁴ while with atelectasis (Fig 4, Series II) likewise partial compensation is observed. In either case one would not expect full compensation since this would remove the chemical stimulus necessary for hyperventilation.

An interesting point may be raised here in connection with oxygen therapy used in the presence of dyspnea during clinical atelectasis. O_2 therapy alone cannot be expected either to completely saturate the arterial blood or to lower the arterial pCO_2 , since both are due to shunt through nonventilated lung. The indicated therapy is re-aeration of the atelectatic lung. Also, as noted previously, if pulmonary tissue containing oxygen is blocked off, or if oxygen ventilation has allowed N_2 to be blown off, a new atelectasis would proceed more rapidly.¹

CONCLUSIONS

1 The contralateral (open) lung ventilation was studied during unilateral atelectasis in dogs.

2 A small increase in total O_2 consumption during atelectasis is probably due to increased cardiac and respiratory work.

3 O_2 consumption data corroborate our previous observations that approximately 50 per cent of the atelectatic lung volume change is reflected by overdistention of the open lung and mediastinal shift and 50 per cent by chest wall and diaphragm shift.

4 The factors effecting the immediate increase in contralateral open lung ventilation during unilateral atelectasis were evaluated. To the previously determined "mechanical effect" of bronchial occlusion is added ventilatory stimulation due to blood chemical changes (arterial O_2 desaturation and increased CO_2). Vagal afferents seem to contribute little to the compensatory ventilation volume.

5 The effects of the pulmonary blood shunt during atelectasis and the changes due to its abolition are described.

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TABIE II SUMMARY OF COLLECTED SERIES OF TRAUMATIC CHYLOTHORAX REPORTED IN THE LITERATURE

COLLECTORS	DATE	NO OF CASES	SEX		AGE	SIDE			TIFRAI Y			MORTALITY
			M	F		R	L	B	THORACOCENT SES ONLY	THORACOCEN TESES FOL LOWED BY LIGA TION OF DUCT	OTIHER	
Shackelford and Fishbein 1935 1936	1938	41	35	6	7 62	21	14	6	20	0	21	50%
Lampson 1936 1948	1948	18	11	7	10 60	11	5	2	7	2	9	40%
Goerwitsch 1918 1954	1954	31	20	11	1/4 64	16	12	2	10	15	6	Entire series—10% Those treated by ligation of duct (15)—0% Those treated other than by ligation of duct (16)—19%

R = Right L = Left B = Bilateral

TABLE III CASES OF TRAUMATIC CHYLOTHORAX REPORTED IN THE LITERATURE SINCE 1948¹

DATE AND AUTHOR	AGE (YR)	SEX	NATURE OF TRAUMA	SIDE OF CHY- LO- THO- RAX	THERAPY	RE- SULTS
1948						
Sundberg ²⁸	36	F	Cough	R	Thoracocentesis only	Rec
Hodge and Bridges ¹¹	49	F	Transthoracic thoraco- lumbar sympathectomy	R	Anastomosis between thoracic duct and azygos vein	Rec
Baldrige and Lewis ¹	35	F	Transthoracic sympa- thectomy	R	Thoracocentesis fol- lowed by ligation of duct in chest	Rec.
1949						
Whiteside et al ³¹	30	M	Bullet wound of chest	R	Thoracocentesis fol- lowed by ligation of duct in chest	Rec
	10	F	Blalock operation	L	Thoracocentesis fol- lowed by ligation of duct in chest	Rec
Goorwitch and Ettleson ⁹	52	M	Extrapleural thoraco- lumbar sympathectomy	L	Thoracocentesis only	Rec *
1950						
Meade et al ²⁰	24	F	Fall down steps	L	Thoracocentesis fol- lowed by ligation of duct in chest	Rec
	35	F	Back diving	?	Catheter drainage only	Rec
Rumel ²³	5	M	Stab wound of left neck	L	Thoracocentesis fol- lowed by ligation of duct in neck	Rec
	?	M	Gunshot wound of chest	L	Thoracocentesis only	Rec
	?	M	Gunshot wound of chest	L	Thoracocentesis only	Rec
1951						
Lowman et al ¹⁸	6	F	Falling piano sounding board	L	Thoracocentesis only	Rec
Frazell et al ⁸	61	F	Left radical neck dis- section	B	Thoracocentesis only	Rec
Shumacker and Moore ²⁷	1/4	M	Blalock operation	R	Thoracocentesis followed by ligation of duct in chest	Rec
	1	M	Blalock operation	L	Thoracocentesis followed by ligation of duct in chest	Rec
1952						
Effler ⁷	1	F	Left thoracotomy and ligation of anomalous subclavian artery	L	Thoracocentesis fol- lowed by catheter drainage	Rec
Hoffman et al ¹²	40	F	Extrapleural thoraco- lumbar sympathectomy	R	Thoracocentesis only	Rec
Wright and Gardner ³⁷	27	M	Automobile accident with crushing injury of chest, dislocation of thoracic spine, and rupture of cord	B	None for chylothorax	D †
Morris and Polk ²¹	34	M	Pistol wound of chest	R	Thoracocentesis fol- lowed by ligation of duct in chest	Rec
Longmore ¹⁷	63	M	Cough	L	Thoracocentesis only	D ‡
Berthrong and Pike ³	40	M	Automobile accident with anterior disloca- tion of thoracic spine	R	Inadequate thoracocen- tesis only	D §
Birt and Connolly ⁴	18	M	Automobile accident	R	Thoracocentesis followed by right phrenic crush	Rec

Fig 1



Fig 2

Fig 1—Roentgenogram two days postinjury demonstrating the left chylopleurothorax and subcutaneous emphysema of the chest and neck (Aug. 28, 1953).

Fig 2—Roentgenogram fourteen days postinjury demonstrating increased amount of fluid with loculation and displacement of the mediastinum (Sept. 9, 1953).

Fig. 4

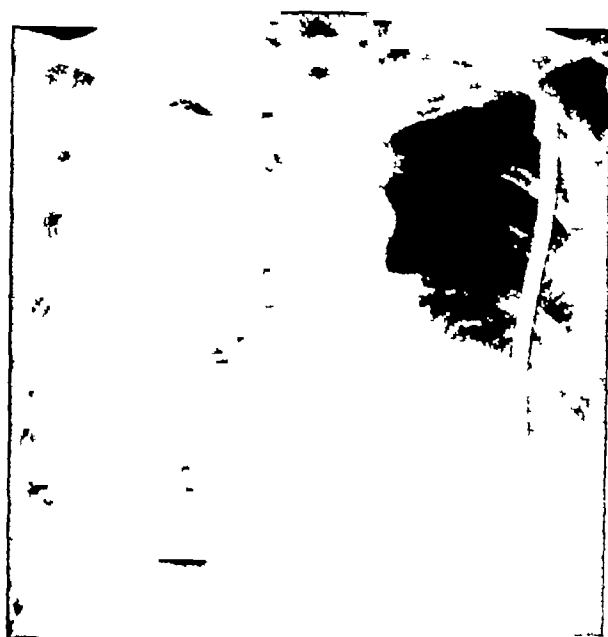


Fig. 5

Fig. 4—Roentgenogram two days postoperatively, demonstrating re-expansion of the left lung and partial return of the mediastinum to normal position (Sept. 14, 1953).

Fig. 5—Roentgenogram three weeks postoperatively, demonstrating complete re-expansion of the left lung and residual pleural thickening at the left base (Oct. 3, 1953).

form of ligation of the thoracic duct if thoracocenteses or catheter drainage for a limited period of time fails to result in sealing of the chylous fistula. Despite the apparent understanding that this subject enjoys currently, two recently published American textbooks on thoracic surgery emphasize the importance of finding the fistulous point in the duct and ligating the severed ends^{10, 29}, one recommends ligation of the duct just above the diaphragm without searching for the point of rupture only for the very debilitated patients so as not to prolong the operative procedure¹⁶, and two others make no mention of the subject at all. As pointed out in a recent article,¹⁴ these findings suggest the incomplete appreciation of the fact that, in order to stop the leakage of chyle from the thoracic duct system above the diaphragm, all that is required is ligation of the duct between the point of leakage and the cisterna chyli, which is not the formidable procedure it is at times described to be.

In selecting the hemithorax through which the lower thoracic duct is to be approached for ligation, it would seem logical to choose the side of the chylothorax,^{15, 16, 29} but not before the following considerations are taken into account. First, there is the greater accessibility of the duct through a right rather than left thoracotomy^{1, 14}, and second, there is the presence or absence of lesions within the chylothorax side for which surgical correction is indicated. Thus, when in the presence of a left chylothorax, repeated thoracocenteses indicate that the lung is expandible and decortication will not be necessary, approach through a right thoracotomy is recommended.¹³ In accordance with these principles, the author's patient was treated by left thoracotomy, and both ligation of the thoracic duct and pulmonary decortication were performed.

Review of the collected series in Table III reveals that expandibility of the lung in chylothorax is satisfactory in the great majority of instances regardless of the method of treatment, provided the leakage of chyle is controlled. The interval between onset of chylothorax and ligation of the duct ranged from zero to eight months and averaged 37 days. In one case, after nine weeks of chylothorax, the lung was found to be encapsulated but without the presence of a definite visceral "peel", and following a difficult decortication re-expansion was incomplete²⁰, this finding was given as one reason for not delaying thoracotomy and ligation of the duct too long after the onset of chylothorax.²⁰ In contrast to this is a case subjected to thoracotomy eight months after onset of chylothorax with the finding that there was no pleural cortex or "peel" and the lung was expandible.¹⁴ The writer's experience conformed entirely with neither of the extreme observations quoted previously, clotting and encapsulation were noted as early as ten days following onset of chylothorax, but the visceral "peel" was very definite at thoracotomy seventeen days after the accident, and decortication was not difficult. In the case of chylothorax in the collected series treated with a single intrapleural instillation of streptokinase-streptodornase for loculation of chyle seventeen days after onset, recovery took place after a few additional thoracocenteses.²⁴

It would seem that in order for chylothorax to complicate a chylous fistula in the neck, traumatic penetration of the parietal pleura would be a pre-

3 Mounting interest in traumatic chylothorax is due both to the safety and success of intrathoracic ligation of the thoracic duct as well as to the increasing frequency of this lesion as a complication of surgical intervention in and about the mediastinum

4 Beginning with Lampson's and including the writer's case, a total of seventeen patients with traumatic chylothorax were subjected to ligation of the thoracic duct. This was followed uniformly by recovery and without unusual morbidity. By contrast, a mortality of 19 per cent was found among the patients in the same series collected by the writer in whom chylothorax was treated other than by ligation of the thoracic duct.

5 The inescapable conclusion is that ligation of the thoracic duct is the treatment of choice when a brief period of management by aspiration or continuous closed drainage demonstrates no tendency for the chylous fistula to seal off spontaneously.

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RECURRENT PLASMACYTOMA OF THE LUNG

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THIS is a follow-up report on a case of plasmacytoma of the mediastinum originally detected by routine chest x-ray in 1944, surgically removed, and reported by us in 1950.¹ Following operation the patient remained under our observation until 1948. He was not seen by us from 1948 until November, 1953, when he returned with symptoms of progressive dyspnea affected by posture especially when lying on his left side. He had observed distention of the neck vessels and was conscious of a loud wheeze in his chest which had increased in intensity over a period of several weeks. Although he dated the onset of his symptoms to August or September, 1953, in retrospect he remembered that he had had a nonproductive cough and wheezing off and on for the preceding year. Because of increasing symptoms he consulted his family physician in November, 1953 and was referred to us for further investigation.

On examination the patient, now 46 years of age, was a well-developed, well-nourished white man. His temperature was normal. There was striking cyanosis of the lips, mucous membranes, and nail beds. A loud inspiratory and expiratory wheeze was heard over the entire chest anteriorly and posteriorly. There were scattered crepitant rales at the left base posteriorly. Respirations were 22, slightly labored. Heart rate was 86, regular. A₂ was greater than P₂. Blood pressure was 174/72. Urinalysis was negative, and blood count was within normal limits with 15.4 Gm hemoglobin, 7,600 leukocytes, and normal differential. X-ray of the chest revealed a large mediastinal mass resulting in pressure and displacing the trachea anteriorly. No evidence of extension or metastasis to the lung parenchyma or pleura was observed. Because of recurrence of a mass at the site of the former tumor, it was decided that surgical exploration should be done without delay.

On Dec. 1, 1953, under general anesthesia the patient was explored by one of us (G. C. A.). When the pleural cavity was entered numerous adhesions were observed along the previous scar. The tumor had recurred and was attached to the lateral arch of the aorta for a distance of 3 inches. The mass was well encapsulated. The tissue was soft in consistency and reddish-brown in color. It was impossible to remove all of the tumor because of its attachment, but as much was removed as could be done safely. The patient withstood the operation well and his postoperative course was uneventful. His breathing was much improved by the third day when he was ambulated. He was ready for discharge

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Fig 1



Fig 3



Fig 4

Fig 3—Posteroanterior roentgenogram Nov 16 1953 reveals large mediastinal mass resulting in pressure displacing the trachea anteriorly.

Fig 4—Rotated roentgenogram Nov 16 1953 reveals displacement of the trachea.

operation, the patient died while climbing into bed following ward exercises. He died about 2 hours to 2 1/2 hours after operation and expired within a few minutes. Autopsy was performed and the cause of sudden death was found to be pulmonary embolism. All gross amount of clots was found to have been removed after operation.

PATHOLOGIC REPORT

Gross. Specimen consists of a fairly well demarcated, irregular, dark red, lobulated mass measuring 4 by 3.5 by 2 cm. This tumor is surrounded by a thin layer of connective tissue. On cross sections, the surface is soft, dark red in color, irregular in shape, and studded with hemorrhagic spots. Accompanying the specimen is an irregular fragment of tissue presenting the same appearance as the primary specimen, and measuring from 1 to 4 by 3.5 by 2 cm.



Fig 6

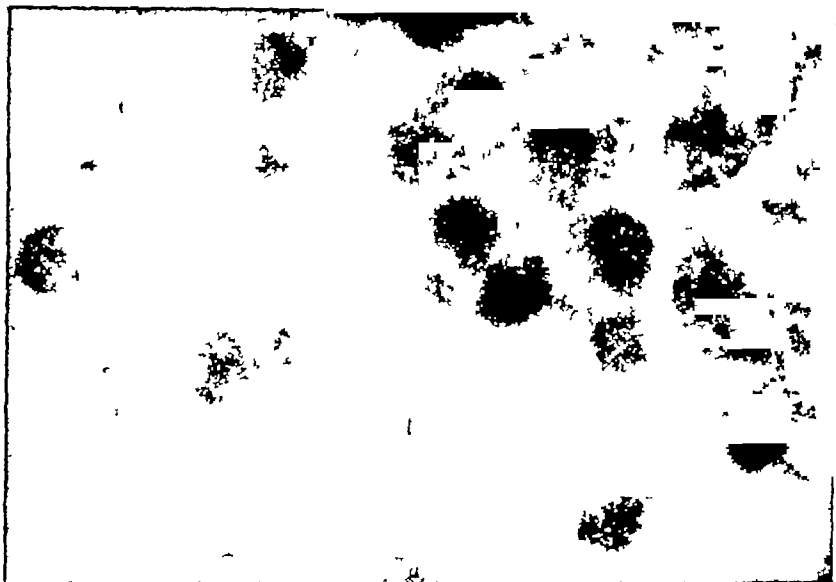


Fig 7

Fig 6—High power photomicrograph showing plasma cells (Approximate magnification $\times 645$ reduced $\frac{1}{2}$.)

Fig 7—Oil immersion view of plasma cells—smear from specimen (Approximate magnification $\times 1475$ reduced $\frac{1}{2}$.)

mediastinum Eggston⁸ has observed plasmacytomas in the upper respiratory tract to become malignant in some instances Hellwig⁹ observed local invasion and distant metastases of plasma cell tumors None of these, however, was in the lung Of eleven cases of plasmacytoma reported by Figi and associates¹⁰ in 1945, five showed frankly malignant behavior with either extensive local invasion or distant metastases Couret,¹¹ in 1946, reported a plasmacytoma of the pyloric portion of the stomach This lesion was locally resected Two months later the patient was explored for obstruction and was found to have large fungating masses throughout the abdomen One month later, autopsy revealed the large, definitely invasive masses to represent plasmacytoma



Fig. 9—Pulmonary infarction causing death

SUMMARY

This is the follow-up on a plasmacytoma of the mediastinum revealing recurrence of the lesion producing symptoms approximately nine years after complete removal of the original tumor, so far as could be determined

In a search of the literature we were unable to find recurrence of such a tumor reported, although extrathoracic plasmacytomas have been observed to result in malignant behavior, invasion, and distant metastases Most plasmacytomas, however, seem to be benign in character

RESECTION AND ANASTOMOSIS OF THE BRONCHUS CARRIED OUT IN A CASE OF BENIGN TUMOR

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THE partial or enuclear excision of minor or major parts of the intrathoracic respiratory tracts, carried out in connection with the various diseases of these tracts, and the reconstruction of the deficiency thus resulting, as well as the substitution of traumatic defects—sometimes bridging over major breaks in the continuity—has been a long-standing ambition of surgery. Ever since operability of pulmonary tumors has been progressing, the problem of reconstructing the respiratory tracts has been acquiring growing prominence. At the same time the therapy of such clinical cases is beginning to develop which up to now—owing to the specific position of the lesion, its delicate topography and, last but not least, the vital function of the respective organs—have seemed surgically insoluble. In these days, when the ever accumulating diagnostic pathologic and chirurgotechnical experiences and our advanced knowledge of pre- and postoperative treatment are all turned to account it is natural that the endeavors of thoracic surgery are not only focused on curing the respective form of disease but also on striving to achieve this aim while maintaining all healthy tissues or parts of the organ. Hence functional-pathologic considerations must prevail in thoracic surgery.

The correctness and justification of this view is proved by our case as herein described.

CASE REPORT

The patient was a 42 year old, well developed and well nourished man, strikingly pale with a complexion indicative of chronic suppuration. His ill health began in 1949, supposedly in connection with pertussis. Since that time he had been suffering from an ever worsening cough in the form of attacks recurring several times daily. In September, 1950, on the occasion of a heavy coughing spell hemoptysis in the amount of 700-800 cc occurred. This was repeated in November, 1951, and in February, 1952. Apart from this, his sputum contained blood four or five times every month. From time to time periods of intermittent fever set in which, after discharge of profuse and sometimes putrid sputum and following penicillin treatment, changed to a subfebrile state. When he was examined by x-ray on such an occasion, the radiologist thought he saw the shadow of a lymph gland in the upper pole of the right hilum. In April 1953 a further very grave pulmonary hemorrhage occurred. In July the patient had repeated pneumonic symptoms, with high fever, then discharge of profuse, putrid, yellowish, and purulent sputum. Because of these symptoms the patient was hospitalized and a bronchoscopy showed a tumor located in the right main bronchus. A biopsy was made and the pathohistologic diagnosis was "oat-cell carcinoma." The patient had lost some weight and was feeling continuous twinges of pain and oppression in his chest, especially during the morning in connection with his recurring bouts of coughing.

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red blood cells, 3,500,000, and of white blood cells, 6,700, the qualitative blood picture apart from a hypochromic anemia of a moderate degree, was normal. Urine was negative. Wassermann reaction was negative. sputum was yellowish, purulent, not putrid, Koch reaction was negative. Blood pressure was 120/80. electrocardiogram showed sinus rhythm.

To save the middle and lower lobes we thought of a bronchoplastic solution. In order to determine the exact topography of the tumor and its relation to its vicinity we carried out a bronchography of the right side which showed a defect in filling the size of about an almond in the lateral wall of the right main bronchus. The tumor originated about 0.5 cm. from the carina and was extending into the intermediate bronchus, occluding the orifice of the bronchus of the upper lobe, so that attempts to fill it with iodized oil were unsuccessful (Fig. 3).

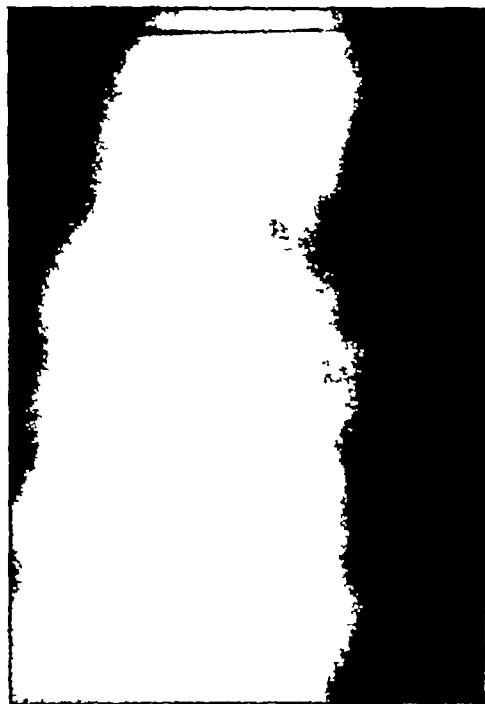


Fig. 2



Fig. 3

Fig. 2—Tomogram. A tumor of the size of a hazelnut located in the bronchus and narrowing the lumen to the width of a straw blade, joined by an extramural shadow.

Fig. 3—Bronchography showing the intrabronchial relations of the tumor. Middle and lower lobes are intact; upper lobe is occluded.

After preliminary introduction of an artificial pneumothorax a thoricotomy was carried out in the sixth intercostal space (Dr. Békfalvi). We found the pleural cavity free. The upper lobe, especially its pectoral segment, was retracted and atelectatic. The fissure of the upper lobe was not developed, so we severed the latter intersegmentally from the middle lobe. After ligating the artery and vein of the upper lobe we isolated the right main bronchus and the intermediate bronchus, exposing them in their entire course, the bronchus of the middle lobe up to its point of origin. On its medial wall we stripped off the pulmonary artery which had been closely connected with it. Then we determined by palpation the place of the tumor. It originated about 0.5 cm. under the carina, occluding the orifice of the bronchus of the upper lobe and running deep down into the intermediate bronchus, about as far as 1 cm. above the point of origin of the bronchus of the middle lobe.

The postoperative course was uneventful. Twenty four hours later the lung expanded, and on the third day we removed the drainage tubes. At the end of the third week the patient left the clinic, fully recovered (Fig 8). On the occasion of the bronchoscopic examinations repeated in the third and the fifth week, we removed the sutures, which were intrabronchially visible, as well as a few growths of granulation tissue that had developed around these. The

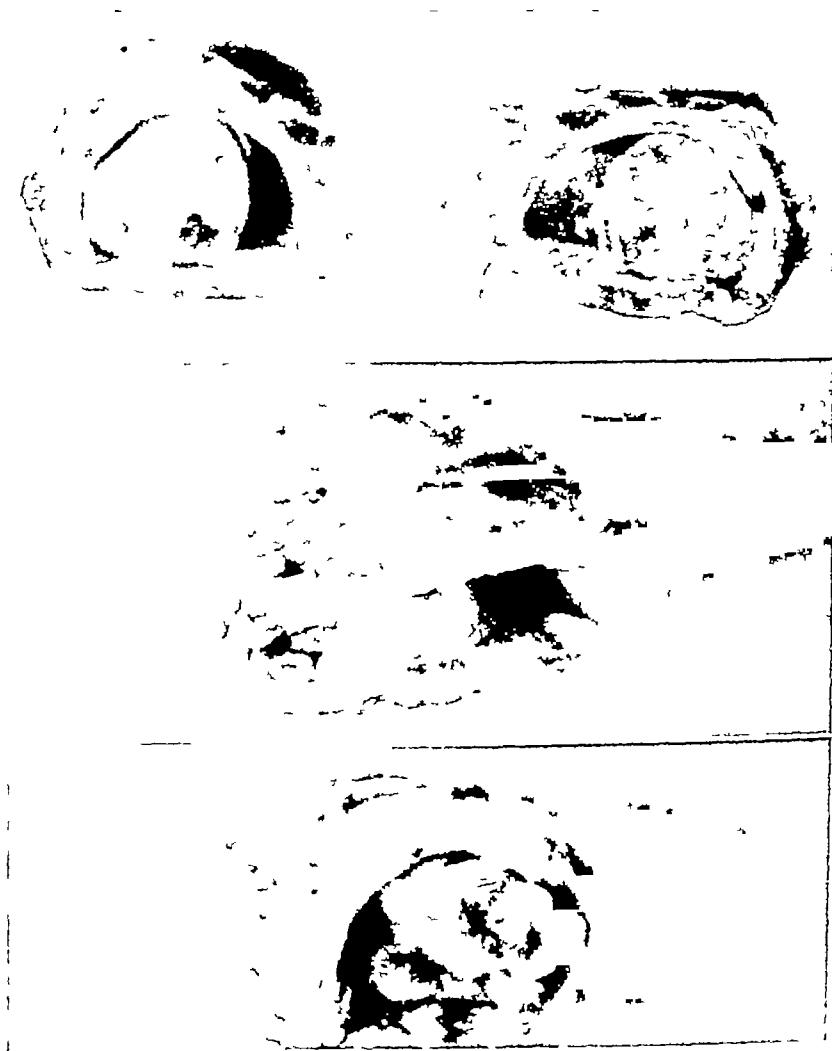


Fig 5—The removed stem and intermedial bronchus. The length of the resected portion is 3 cm.

lumen of the bronchus was clear. A bronchography made in the sixth week did not show any stenosis at the site of the anastomosis. In the course of the expansion, the middle lobe had shifted upward (Fig 9). Six months later, the patient was free from complaints and going about his former work.



FIGURE 1. PRONCHUS (10X)

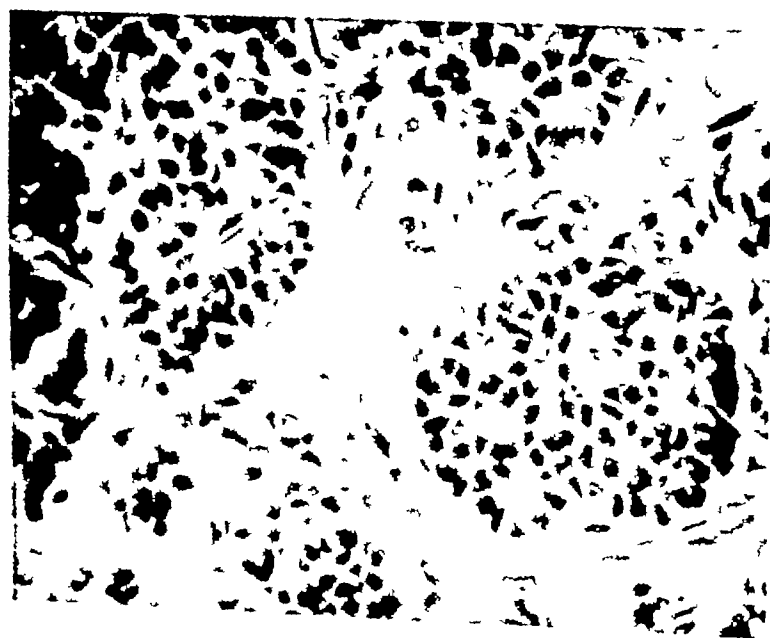




Fig 8—Postoperative roentgenography of the thorax. The remaining lobes are fully expanded and show normal ventilating conditions.



Fig 9—Postoperative bronchography. There is no stenosis on the place of the anastomosis. In the course of the re-expansion the bronchus of the middle lobe has shifted upward, indicating that this lobe has taken the place of the removed upper lobe.

DISCUSSION

Without this surgical solution, the standard operation (a pneumonectomy) would have been unavoidable, owing to the delicate position of the tumor. A bronchoscopic removal was out of the question, partly because of the uncertainty of the initial diagnosis and the fact that the tumor was attached to the wall of the bronchus on a broad basis partly because of the patient's extraordinary tendency to hemorrhage, and last but not least, because of the so called "potential malignancy" of adenomas which has been discussed in numerous journals and proved by several authors. A removal by way of bronchotomy could not be carried out because of the size of the tumor and its extramural relations. Furthermore, in cases of tumors carrying the possibility of malignancy, this form of treatment cannot be considered a radical one owing to the character of the intervention.

In our case, by totally resecting the right main bronchus and the intermediate bronchus and bridging over the resulting break by an end-to-end anastomosis, we succeeded in saving the intact and soundly functioning middle and lower lobes and were able to avoid pneumonectomy, which always means mutilation.

Our case further proves the possibility of removing a considerable segment of one of the main bronchi and uniting it without using an inserted medium (prosthesis or grafts), by exposing and mobilizing the bronchus on a maximum section. Contrary to the sparse data in the literature published so far, according to which the maximum range of resection at the bronchus covers a portion of 2 cm, the removed section of the bronchus, in our case, measured 3 cm.

The preoperative conditions for a successful intervention are as follows:

- 1 Strict asepsis. An infection causes insufficiency of suture, while healing by second intention effects a proliferation of tissue, which always leads to cicatrization and stenosis.

- 2 An airtight suture line. Its lack prevents expansion and involves the well-known inherent dangers.

- 3 Exact joining. This is necessary in order to achieve airtight closing and uniformity of caliber.

- 4 Avoiding overtautness. This may be achieved by proper mobilization of the various elements, a taut suture line may bring about dehiscence.

- 5 An anastomosis free from stenosis. Without this we might imperil the success of the intervention.

- 6 Re-epithelization. This needs time, and the foreign-body granulomas developing in the suture line may temporarily interfere with it.

- 7 Avoiding a stasis of secretion. It is necessary to ensure ventilation so as to prevent infection of the distal lung area.

- 8 Achievement of quick expansion of the lung. If possible, within the first twenty-four hours.

- 9 Adequate suture material. Because of the tautness which, to a certain degree, is unavoidable, it seems more advisable to use nonabsorbable suture.

materials, though these are always associated with foreign-body reactions appearing in the form of granulation tissue. For that reason the endoscopic removal of the sutures should be effected as soon as possible, e.g., at the end of the third week.

10 It is necessary to cooperate closely with the bronchologist so as to obtain a precise topographic and pathohistologic diagnosis, to clear up the relations of the lesion and to let the patient have the exact pre- and postoperative treatment required.

SUMMARY

The indications for applying bronchus resection with anastomosis and plastic operation are as follows: (1) lesions of the respiratory tracts—ruptures, fractures, or operative lesions, (2) circumscribed tumors of the respiratory tracts, (3) specific and nonspecific stenoses of the tracheobronchial tree, (4) space-consuming processes occurring outside the respiratory tract, in most cases tumors which, because of their close contiguity to these tracts, could otherwise not be removed, that is to say, the surgical solution of which could only be accomplished by removing part of the respiratory tract itself, (5) functional indication to confine the resection strictly to the portion of the lung affected or injured.

As regards bronchus resection and end-to-end anastomosis carried out on human beings we have reports by Gebauer^{2, 3} on several such cases, and single case reports by D'Abreu and MacHale,¹ Mathey and Oustrieres,⁵ Métras and co-workers,⁶ Paulson,⁷ Scannel,⁸ Kinsella, and Johnsrud,⁴ and Weisel and Jake.⁹

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TOLERANCE OF BRONCHIAL WALL TO HIGH LEVEL DOSAGE OF RADIOACTIVE COLLOIDAL GOLD FOLLOWING INTRABRONCHIAL INJECTION

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IT HAS been demonstrated that when radioactive colloidal gold is injected into the submucosa of the bronchus of dogs, the colloidal sol is transported via the lymphatics from the site of injection to the regional lymph nodes where it is trapped and concentrated¹. Appreciable concentration exists in the lymph nodes within four hours after injection². In some sixty dogs receiving 2 to 3 mc. of Au^{195} by this method, no untoward effects have been noted. Since this method of delivering radiation has suggested the possibility of offering palliation to patients with inoperable bronchiogenic carcinoma, we have outlined the following experiment to determine the tolerance of the bronchial wall to high level dosage of Au^{195} of the magnitude which conceivably might be required to have therapeutic effect on bronchiogenic carcinoma.

MATERIAL AND METHODS

Radioactive colloidal Au^{195} with particle size ranging from 0.003 to 0.004 micron was used*. The isotope has a half life of 2.7 days. It emits beta rays with maximum energy of 0.97 Mev and gamma rays with energy of 0.411 Mev. Fifty per cent of the beta rays penetrate only 0.38 mm. and are the primary source of radiation effect in localized areas. The gamma rays penetrate deeply and are useful for external counting but they may also contribute to both local and whole body radiation.

The isotope was injected into the intermediate bronchus of nine dogs by the following technique. Under intravenous Nembutal anesthesia, a 7-10 bronchoscope was introduced directly into the trachea and advanced to the selected site for injection. The distal end of the bronchoscope was then angled slightly toward the proposed site of injection to bring this region under minimal tension. A 45 cm. needle of the type used for injection of esophageal varices (Pilling Br. 800) was introduced through the bronchoscope under direct vision. The tip of the needle was inserted through the taut bronchial mucosa for a distance of about 3 mm. A syringe containing the radioactive

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gold was connected with the needle and the injection made. If the needle is in the proper submucosal plane, the injection should require very little force. After the injection, the needle was immediately removed. Occasionally a drop of blood could be seen on inspection of the site of injection.

The two common errors in inserting the needle are allowing the tip to enter, or impinge on, a cartilaginous ring, or passing the needle through the entire thickness of the bronchus into a bronchial or a pulmonary vessel. Should the cartilage be entered or impinged on by the tip of the needle, considerable resistance will be met to advancement of the needle and to the injection of fluid. If a major vessel is entered, often blood will leak back through the needle. Blood will not be seen to drip from the hub of the needle if the tip is in the proper submucosal plane.

Two dogs received 30 mc of Au^{198} diluted in 2 c c of normal saline solution. Three dogs received 50 mc and one dog 68 mc in similar dilution. One dog received 75 mc diluted in 2.6 c c and one dog received 100 mc of Au^{198} in 3.45 c c. All animals survived and appeared to be in a normal state of health at the time of autopsy. They were autopsied from twenty-one to sixty-six days after injection. Histologic sections were made of the site of injection, the lung, lymph nodes, bone marrow, spleen, liver, and kidneys.

RESULTS

One animal which had received 50 mc had pericarditis and mediastinitis resulting from leakage of the radiogold into the mediastinum. It is probable that the needle passed through the bronchial wall since no local effect could be found in multiple histologic sections of the bronchus, and no change in the lymph nodes attributable to radiation was found. The dog receiving 85 mc had some aspiration into the lower lobe of the right lung, but showed considerable lymph node change in addition to the radiation effect on the lung tissue. These two failures represent errors in technique and point out definite dangers in the therapeutic application of the method. However, it might be added that to our knowledge they are the only technical failures that have occurred in sixty animals we have injected in this manner.

The remaining seven animals showed no gross evidence of damage to the bronchial wall at the site of injection (Fig 1). There was, however, microscopic change in the vessels of the bronchial wall attributable to radiation. The damage was never severe and the wall remained intact (Fig 2). Bone marrow from ribs and sternum was within normal limits as compared with control animals. There were no changes attributable to radiation in the lungs, liver, spleen or kidneys. Multiple sections were made of the upper tracheal, right and left paratracheal and right and left subcarinal lymph nodes. Varying degrees of radiation change occurred in these nodes in all animals. They ranged from complete necrosis of all the above lymph nodes through isolated areas of focal necrosis to dense scars in portions of the nodes (Figs 3, 4, and 5). This patchy distribution of radiation effect could not be attributed solely to the dosage of isotope used, site of injection, or time



Fig 1—Lung of dog receiving 50 mc of Au^{198} . There is no visible local effect.

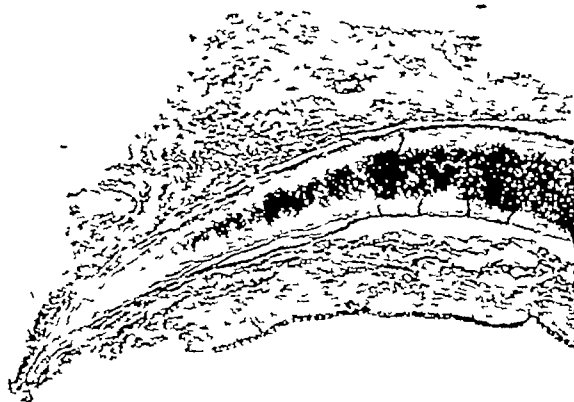


Fig 2—Section showing intact bronchial wall at the site of injection. There is slight local radiation reaction in the blood vessels.

interval between injection and autopsy. The possibility that lymph en culation throughout any given node might be impaned by previous infection was suggested in one animal that had a granulomatous lymph noditis presumably secondary to histoplasmosis of the lung. This of course also suggests that in a lymph node containing metastatic cancer, the isotope might not reach effective concentration. This problem is now under investigation.

Fig. 3



Fig. 4

Fig. 3—Low-power magnification of right paratracheal lymph node. There is marked radiation necrosis with only a small amount of residual lymphoid tissue seen in the lower portion of the photomicrograph. This dog had received 50 mc. of Au^{198} five weeks previously.

Fig. 4—High power magnification of lymph node shown in Fig. 3.

SUMMARY AND CONCLUSIONS

A colloidal solution of Au^{198} was injected into the submucosa of the intermediate bronchus of nine dogs. Histologic examination of the site of injection, regional lymph nodes, lungs, liver, spleen, kidneys, and bone marrow was made.

Evidence of radiation effect was confined to the injection site and its lymph drainage area

Technical errors in injection resulted in pericarditis and mediastinitis in one animal and radiation necrosis in the lower lobe of the right lung in another

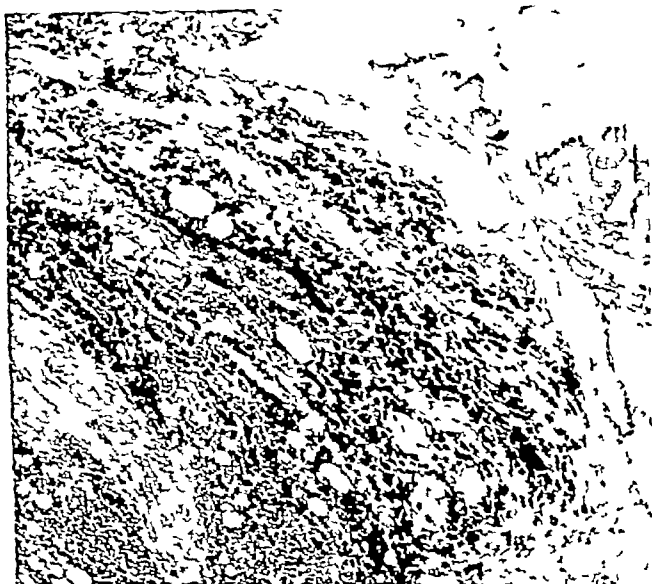


Fig 5—Photomicrograph of lymph node showing deposition of gold and radiation scar sixty-six days after injection of 30 mc. of Au^{198} into the bronchus

There were no untoward effects at the site of injection, using relatively high dosages ranging from 30 to 100 mc, indicating that mobilization from the mucosa to the nodes takes place rapidly before severe local injury results

Radiation effect of varying degree was present in the lymph nodes of all animals

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INTRABRONCHIAL LEIOMYOMA

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PRIMARY muscle neoplasms of the lung are infrequent. In the lung, as in other organs, the muscle tumors are leiomyoma, leiomyosarcoma, rhabdomyoma, and rhabdomyosarcoma. These various muscle tumors of the lung have been described in the literature in detail, and an analysis of the available cases is summarized in Table I. Of the recorded leiomyomas two presented as intra-bronchial tumors of definite bronchiogenic origin, and six were parenchymal in location. We believe the present case is of interest primarily because, to the best of our knowledge, it is the third intrabronchial leiomyoma to be reported, previous cases having been reported by Turkington and associates¹ and Freireich and co-workers.² The cases by Brahdy,³ Foikel,⁴ and Fianco⁵ were clearly pulmonary leiomyomas, but it is unfortunate that the exact sites of origin and statements as to intra- or extraluminal extension are lacking. The case reported by Williams and Daniel⁶ was a leiomyoma surrounding and obliterating the left lower lobe bronchus. The case reported by Deussing⁷ is cited as the second one in the literature. We do not believe that his case represented benign leiomyomas (see discussion below).

REPORT OF A CASE

A 43 year old Negro woman was admitted to the Emergency Room of the St. Philip Hospital, Medical College of Virginia, on Sept. 6, 1952, with the history of sudden loss of consciousness and convulsion. The patient was first seen at a hospital of a neighboring city, at which time she was having continuous generalized convulsions. She was subsequently referred to this hospital after emergency treatment. There was no history of past illnesses except for the removal of an abdominal tumor eight years previously. Her family history was not unusual. There was no history of known hypertension. Physical examination on admission revealed a blood pressure of 200/150, pulse 140, respiration irregular with periods of apnea necessitating artificial respiration. The skin was cool and of good turgor. The patient was completely unresponsive. There was no nuchal rigidity. The pupils were unequal, with the left quite small. The optic disc was blurred on the right, with the vessels full and tortuous. The left optic fundus was not adequately visualized. The heart was enlarged with a rapid but regular rate. No murmurs were heard. The upper respiratory tract was filled with vomitus. The lung fields contained many râles. The abdomen showed a low, midline, healed abdominal scar. Masses or organs were not palpable. The external genitalia were not unusual. All the extremities were flaccid with reflexes absent in the lower extremities, but active in the upper. Positive Babinski reflex or ankle clonus was not elicited. The cranial nerves were difficult to evaluate adequately. The patient was given supportive therapy to no avail, and she expired one and one-half hours after arrival at this hospital.

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Fig 1—Gross autopsy specimen showing origin within right upper lobe bronchus at bifurcation from main-stem bronchus



Fig 2—Low-power photomicrograph of microscopic slide revealing insertion of leiomyoma between bronchial cartilages ($\times 75$ reduced 14.)

TABLE I

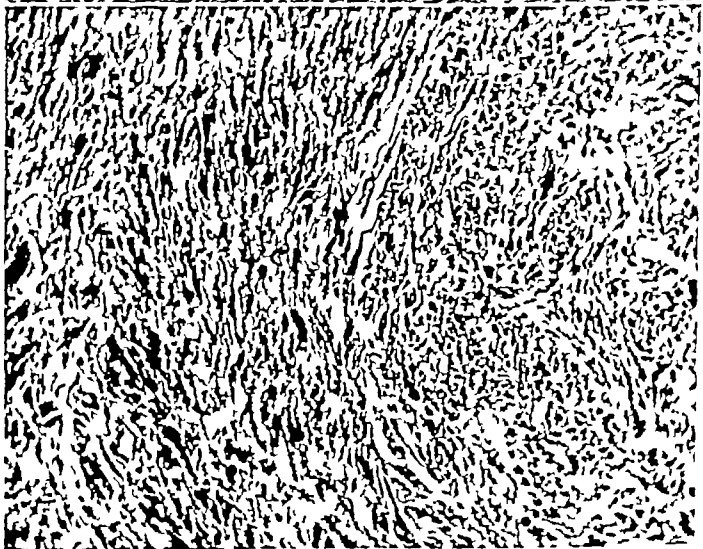
AUTHOR	YEAR REPORTED	SEX	AGE (YR.)	TYPE OF SPECIMEN	LOCATION	DIAGNOSIS
Holbregg ¹¹	1898	M	23	Autopsy	Left lung	Rhabdomyoma
Zipkin ¹²	1907	F	[Stillborn, 1,800 grams]	Autopsy	Left lung	Congenital adenomatoid myoma
Lorkel ¹⁴	1910	F	63	Autopsy	Left upper lobe	Leiomyosarcoma
Francis ¹⁵	1929	F	56	Autopsy	Right upper lobe	Leiomyoma
Richardson ¹³	1929	M	39	Autopsy	Right upper lobe (most probable)	Rhabdomyosarcoma
Neumann ⁹	1938	F	66	Autopsy	Left lower lobe	Leiomyosarcoma
McDonald and Heather ¹⁴	1939	M	52	Autopsy	Right upper lobe	Rhabdomyoma sarcoma toties
Brunn and Goldmans	1940	M	51	Autopsy	Left upper lobe	Leiomyosarcoma
Bradley ³	1941	F	18	Surgical specimen	Right lower lobe	Fibroleiomyoma
Randall and Bladsgo	1946	M	34	Bronchoscopic washings and autopsy	Right upper lobe	Leiomyosarcoma
Kraus et al. ¹⁰	1948	M	48	Biopsy and surgical specimen	Right upper and right middle lobes	Myoblastoma
Holinger et al. ¹	1950	F	6	Surgical specimen (tissue from bronchi)	Right lung, no exact localization	Myosarcoma
Turkington et al. ¹	1950	F	61	Surgical specimen, pneumonectomy	Left lower lobe bronchus	Leiomyoma
Williams and Daniels ⁶	1950	F	8	Surgical specimen (pneumonectomy)	Left lower lobe	Leiomyoma
Freureich et al. ²	1951	M	61	Surgical specimen, pneumonectomy	Right main stem bronchus	Leiomyoma
Mishkin ¹⁵	1951	F	65	Autopsy	Left upper lobe	Myosarcoma
Johnson et al. ¹⁰	1952	M	52	Surgical specimen (pneumonectomy)	Right upper lobe	Leiomyosarcoma

Watson and Amlund ¹⁰	1954	M	57	Surgical specimen (lobectomy)	Right middle lobe	Leiomyosarcoma
		F	63	Surgical specimen	Left upper lobe	Leiomyosarcoma
		F	66	Surgical biopsy	Left lower lobe	Spindle-cell tumor— myosarcoma
		M	66	Surgical specimen (local removal)	Right middle lobe	Leiomyosarcoma
		M	4	Autopsy specimen	Right main stem bron- chus	Leiomyosarcoma
		F	67	Bronchoscopic biopsy	Left lower bronchus	Myosarcoma

Gross Post Mortem Findings—A massive pontine hemorrhage was found with no evidence of an aneurysm being demonstrated. All clinical findings could be related to this lesion.

An incidental finding was a grayish white, firm, bronchial "adenoma like" nodule, situated within the lumen of the right upper lobe bronchus, just distal to its origin from the

A



B

Fig. 3—Bundles of spindle shaped cells on (A) longitudinal and (B) cross section ($\times 200$ reduced $1\frac{1}{2}$.)

right main stem bronchus (Fig. 1). The nodule did not cause complete obstruction of the bronchus, and the lung parenchyma distal to this lesion gave no evidence of abscesses or bronchiectasis. It measured 1.0 cm. in diameter and was connected to the wall of the

bronchus through a base measuring 0.7 cm. On cut section, the nodule was of homogeneous gray color, without subsidiary nodulations, striations, or markings. Gross examination of a microscopic section revealed the base of the nodule to be inserted between two segments of the bronchial cartilage.

Microscopic Description—The tumor appeared to have arisen from bronchial musculature and was interposed for a short distance between two bronchial cartilages, thereby separating the latter (Fig. 2). It extended for a short distance between the cartilages but did not show the deep penetration of the bronchial wall which so frequently characterizes bronchial adenomas. In a number of sections examined there was no invasion of regional lymph nodes. At the base of the tumor, several large dilated bronchial glands filled with mucus were seen. The neoplasm was characterized by whorls of interlacing bundles of spindlelike cells (Fig. 3).

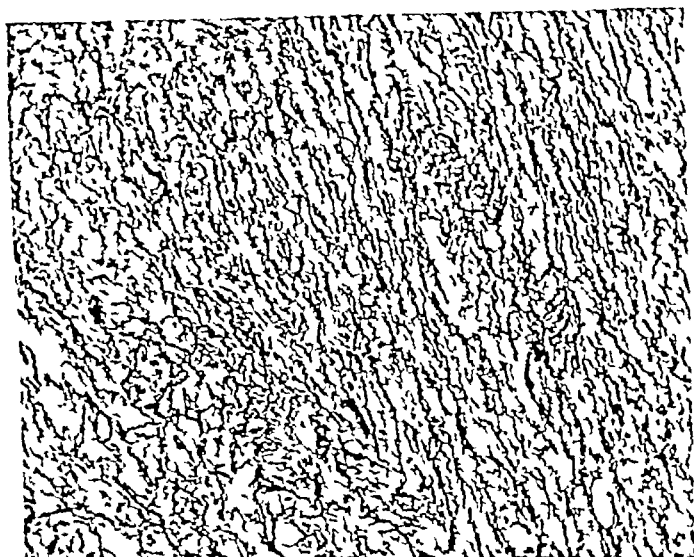


Fig. 4—Reticulum stain demonstrating fine network of reticulin oriented to the long axis of muscle fibers. ($\times 200$ reduced $\frac{1}{2}$.)

Examination of hematoxylin and eosin preparations showed that the cells were characterized by clumps of ovoid nuclei, devoid of nucleoli, atypical mitotic figures, or abundant chromatin content. The cytoplasm was abundant and "streamed out" from the nucleus, identical to the appearance seen in a fibrocyte or myocyte. When the intensity of the microscopic light was diminished, longitudinal striations became apparent. A careful search failed to reveal the presence of cross striations. No necrosis was found. As the tumor grew into the lumen of the bronchus, it carried a covering of respiratory epithelium. This epithelium showed no tendency toward squamous metaplasia, as is frequently the case in benign intraluminal bronchial neoplasms. A Masson trichrome stain revealed abundant blue staining collagen running in between the smooth muscle fibers. A phosphotungstic acid hematoxylin preparation readily selected blue staining strands of smooth muscle fibers with their longitudinal striations. Sections stained with Verhoeff and van Gieson stains surprisingly revealed most tumor stained yellow, a common finding already referred to by Zipkin.¹² A fat stain did not show any sudanophilic material. A Wilder periodic acid Schiff stain demonstrated a fine network of reticulin oriented to the long axis of the muscle fibers (Fig. 4). No aldehyde was demonstrated.

DISCUSSION

The importance of recognition of an intrabronchial leiomyoma lies in the necessity of differentiating this lesion from other intrabronchial neoplasms during bronchoscopy. The more common lesions to be differentiated from intrabronchial leiomyomas are intrabronchial adenoma, fibroma, chondroma and



Fig. 5—Gross specimen of bronchial adenoma

malignant neoplasms. Treatment may vary with a given type of lesion, with subsequent difference in recurrence, adequacy of removal, morbidity, and, possibly, mortality. On gross inspection of these lesions, this cannot be readily done owing to the similarity of their appearances, as evidenced by the accompanying photographs (Figs. 5 and 6). Contrariwise, microscopically there is usually little diffi-

culty in establishing the diagnosis. Difficulty may on occasion arise in the microscopic diagnosis of intraluminal bronchiogenic tumors, for muscle tissue may form a component of parenchymal hamartoma, and likewise collagen may be found in leiomyomas. With use of the van Gieson stain, a leiomyoma may appear to be composed of pure myocytes, but with other differential stains such as the Masson trichrome, and phosphotungstic acid hematoxylin, the collagenous component will be readily apparent. This lack of staining of collagen with the van Gieson stain may possibly be due to the relative immaturity of the collagenous portion of the tumor. Morphologically, the site of origin of the presented leiomyoma corresponded to the smooth muscle of the bronchial wall. However, origin from smooth muscle of a blood vessel cannot be excluded.



Fig. 6—Gross specimen of bronchial hamartoma.

The tumor presented here had as a prominent feature considerable numbers of fibrocytes, as demonstrated by Masson trichrome stain. On the other hand, fibroblasts may play a prominent role in the make-up of hamartomas of the lung. We believe that the connective tissue of the tumor herein reported is a response to the leiomyomatous proliferation, rather than a true neoplastic component of the tumor. However, it is always difficult to decide whether tissue such as this constitutes reactive hyperplasia or neoplasia. Hence, some may regard the tumor presented here as a fibroleiomyoma. The analogy lies in the leiomyomas of the uterine musculature where fibroblasts are easily demonstrated.

The case of multiple primary myomas of the lung described by Deussing in 1912 has been handed down from one article to another in the English language

as an example of a benign tumor^{2, 3, 10} Fiebreich,² for example, includes Deussing's report in a list comprising leiomyoma arising from smooth muscle and limited to the bronchus. From Deussing's findings, which incidentally were not illustrated, it is clear that the invasion of the pulmonary vein with extension into the left auricle is not in keeping with the present concept of the behavior of a benign tumor. Furthermore, one of the tumors contained an "epithelial glandular complex." There is no mention of the presence or absence of mitoses in any of the tumors described.

The report of the cases of rhabdomyoma (teratomatous),^{11, 12} and its malignant counterpart, rhabdomyosarcoma, lends itself well to the doctrine of totipotentialities of the myoblast. The occurrence of these striated muscle tumors could be maintained on the basis of the probability of the myoblast differentiating into striated muscle. The diagnosis of a highly neoplastic muscle tumor of smooth and striated components is at times in doubt, due to the difficulties in differentiating them from a highly anaplastic primary bronchogenic carcinoma.

To date two cases of pulmonary glandular cell myoblastomas have appeared in the English literature.^{15, 16} On bronchoscopy, both presented as an intra bronchial mass. At the present time the histogenesis of benign granular myoblastoma is unsettled. Various theories have been proposed based on a degenerative process, origin from muscle, and origin from nerve fibers. This lesion is not to be confused with muscle neoplasms of the lung such as the leiomyoma presented in this case, which represents a true neoplasm of smooth muscle, most probably of bronchial origin.

SUMMARY

A case of intrabronchial leiomyoma with a gross appearance indistinguishable from adenoma and fibroma of the larger bronchi is presented. Difficulties in diagnosis are discussed.

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THE MASKED BRONCHOPLEURAL FISTULA

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THE symptoms and signs of postresection bronchopleural fistulas are usually definite and unequivocal. Occasionally, however, these fistulas develop in such a manner as to make their presence unsuspected until a catastrophe supervenes. Undoubtedly many such lesions heal spontaneously without having been definitely diagnosed as a complication.

Failure to detect such a fistula might occur in patients who are referred for operation to surgical centers and then returned to a sanatorium for postoperative care. A similar situation might develop in small tuberculosis hospitals where busy consultant surgeons must leave the postoperative care to staff members who are not conscious of the dangers that lurks in the "alveolar leak," "space defect," "reluctant re-expansion," or any of the other numerous euphemistic terms which are used to describe this complication. The variation in frequency of such complications reported by many surgeons may reflect the zeal with which the search for confirmatory evidence of the presence of these fistulas is pressed.

We have selected the term "masked fistula" to describe the lesions which furnish unquestioned evidence of their presence after a period in which the patient seemed to be progressing satisfactorily. Some of them have had a rare evidence of an apical aneurysm, but all have had in common the factor of apparent favorable progress prior to the appearance of definite signs of fistula.

Resection has displaced thoracoplasty as the most commonly used major surgical procedure in the therapy of pulmonary tuberculosis. As surgical techniques were improved and anatomical knowledge of the lung structure increased, it became possible to remove smaller and smaller segments of the lung. This is well illustrated in the reports submitted to the Thirteen Veterans Administration Chemotherapy Conference in February, 1953 (Fig. 1). It was shown that from October, 1947, to October, 1948, 175 resections were reported. Pneumonectomies accounted for 45.1 per cent, and 54.9 per cent were lobectomies. No segmental resections were done. From January, 1951, to January, 1953, 818 resections were reported. Ten and eight-tenths (10.8) per cent of these were pneumonectomies, 32.1 per cent lobectomies, and 57.1 per cent wedge and segmental resections. From January, 1953, to January, 1954, 1,235 resections were reported. Of these 4.9 per cent were pneumonectomies, 29.9 per cent lobectomies, and 65.1 per cent segmental resections. With the increase in segmental resections, the possibility of smaller alveolar air leaks is augmented.

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This increase in benign air leaks is also accompanied by an increase in the number of true fistulae and imposes upon us the responsibility of carefully screening these lesions so that a proper surgical attack may be carried out

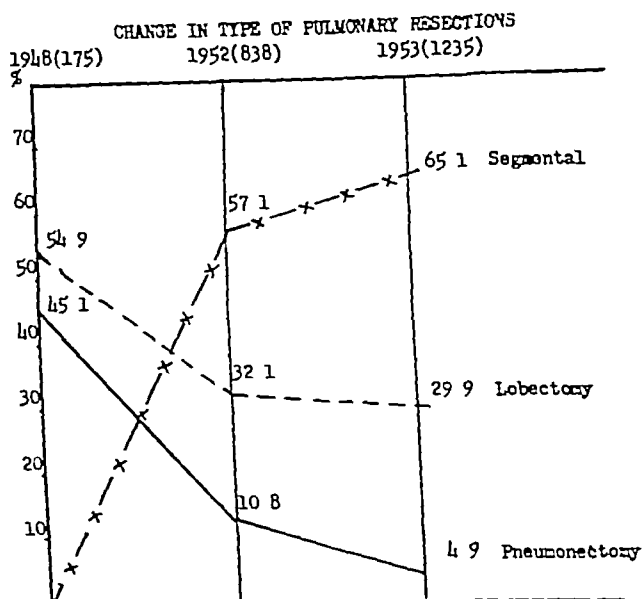


Fig 1

At Oteen, 625 resections were performed between Oct 1, 1945, and Sept 1, 1953 (Table I). Twenty-four, or 3.83 per cent, of the 625 patients who had undergone a resection developed what we term a masked fistula. A breakdown of the 24 fistula cases reveals the fact that 12 followed pneumonectomy, 3 followed lobectomy plus a wedge resection, 3 followed lobectomy, and 6 followed segmental resection. The diagnostic criteria for such lesions plus several case histories will be presented in the balance of this report.

TABLE I

	PNEUMONECTOMIES	LOBECTOMIES	SEGMENTALS OR WEDGES
Tuberculous	172	184	158
Nontuberculous	35	67	9
Total	207	251	167

SYMPTOMS AND SIGNS

The following symptoms and signs are those which have been most helpful to us in suspecting or diagnosing a "masked fistula."

Subjective Symptoms—

- 1 Pain or tightness in the chest
- 2 Sensation of "blowing air" in the pleural cavity
- 3 Cough—increased when lying on nonoperated side and productive of reddish or brown-tinged sputum.

Objective Signs —

- 1 Return of fever after a postoperative return to normal
- 2 X-ray evidence of an unabsorbed or increased amount of intrapleural air, or the appearance of an air pocket in a new location
- 3 Unexplained lowering of the intrapleural fluid level
- 4 Increase in bloody sputum or fluid expectoration after instillation of a fibrinolytic agent such as varidase
- 5 Expectoration of dyes, such as methylene blue, following instillation into the hemithorax or packing in a sinus tract
- 6 Smell or taste following instillation of an odor-producing material such as ether into the pleural cavity
- 7 Bronchoscopic visualization of a fistula
- 8 Bronchographic demonstration of an abnormal communication between the bronchial tree and the pleural cavity
- 9 Retrograde entrance of radiopaque materials in the bronchial tree after injection into a chest wall sinus
- 10 Intermittent or persistent subcutaneous emphysema
- 11 Demonstration of an inexhaustible "air pocket" on thoracentesis
- 12 Sudden appearance of a pneumothorax after re-expansion of the lung
- 13 Failure of the remaining lung, if any, to re-expand on the operative side

CASE HISTORIES

CASE 1—H H, a white man, aged 28 years, had had a diagnosis of far advanced pulmonary tuberculosis established in 1947. When admitted on Oct 1, 1951, a tension cavity with an adjacent 2 cm caseous nodule was present in the right upper lobe. The tension cavity closed under pneumoperitoneum and chemotherapy, but the caseous nodule persisted and an apical posterior segmental resection was done, July 10, 1952. Postoperatively there was a low grade daily elevation of temperature until the twenty-fourth day when a sudden rise to 103° F occurred. This subsided to normal in one week. X-ray films revealed an apical air space about 4 by 3 by 2 cm in size on the first postoperative day. This persisted and on the twentieth day a fluid level was present for the first time. When a needle was inserted, an unlimited amount of air could be removed. Varidase was instilled and produced an immediate cough and a complaint of a salty taste in the mouth. On Sept. 12, 1952, a four rib thoracoplasty was performed, and the subsequent course was uneventful.

A diagnosis of bronchopleural fistula was established by fulfilling at least four of our criteria. X-ray appearance was rather benign, and this case might have been classified as an alveolar leak corrected by a thoracoplasty with a favorable effect upon our morbidity statistics.

CASE 2—W E N, a 33 year old white man, was given a diagnosis of pulmonary tuberculosis in 1946. In October, 1950, far advanced bilateral disease with cavitation in the right hilar area was present. He was treated with bed rest, pneumoperitoneum, and chemotherapy until Feb 7, 1952, when a right lower lobectomy plus wedge resection of nodules in the middle lobe and apical segment of the upper were done. Postoperatively the remaining lung quickly re-expanded and filled the pleural cavity. Drainage tubes were removed on the second day. On the fourth postoperative day x-ray films revealed a 50 per cent pneumothorax on the right. Air was removed by needle and the lung was re-expanded on the eighth postoperative day. The temperature reached normal on the twelfth day. Acid fast organisms persisted in the sputum. A five rib protective thoracoplasty was done, March 10, 1952. An intermittent positive sputum was present thereafter, but his course was otherwise satisfactory.

On Mar 3 1952, three black silk sutures were coughed up. Bronchoscopic examination on June 23, 1952 revealed an apparent opening in the lower lobe bronchial stump. A positive sputum persisted in spite of treatment with Isoniazid, and on July 25, 1952, the remainder of the right lung was resected. A definite fistula was present, opening into a small abscess pocket between the lower lobe bronchial stump and the right margin of the esophagus. His subsequent course was uneventful.

A bronchopleural fistula was not suspected in this patient until a bronchoscopic examination was done four and one half months after the initial resection. In spite of the paucity of symptoms a walled-off abscess was present which was responsible for the continued elimination of acid fast organisms.

CASE 3—S C S, a 34 year old white man was admitted Sept 6 1946 for treatment of pulmonary tuberculosis with cavitation of the left upper lobe. Treatment included a left phrenic crush in October, 1946 pneumoperitoneum and streptomycin. Following this, a left six rib, two-stage thoracoplasty was done in September 1948. On Jan 18, 1949, a left upper lobectomy was done. Postoperatively his course was satisfactory until a minute pleurocutaneous (supposedly) sinus was noted on Feb 7 1949. This persisted without fever and with minimal drainage. On April 9, 1949 (3 months postoperatively), a persistent slightly productive cough was noted. On May 5 1949 the small sinus tract was explored and a cavity found in the pleural space area. No bronchopleural fistula was noted, however Bronchoscopies had been normal April 5 and May 19 1949. It was noted that on May 20 1949 Chloresium used on the wound packing was coughed up in the sputum. He remained asymptomatic thereafter and his wound partially healed. Because of the persistent space, a partial scapulectomy and rib resection was done June 1 1949. A sinus tract persisted with no symptoms. On Dec 13 1949 the patient stated he was blowing air through his sinus. On Dec 15, 1949, methylene blue instilled into the sinus tract was not coughed up in the sputum, but, when this was repeated Dec 31 1949, the dye was at once expectorated on coughing. Bronchogram on Jan 6 1950 showed no bronchopleural fistula. On Feb 15 1950 a third stage thoracoplasty was done with resection of ribs 10 and 11. Revision of the sinus tract was done on March 17, 1950. Bronchoscopy Mar 18, 1950 showed for the first time a small bronchopleural fistula. On Sept 29, 1950, another Schede thoracoplasty was done and the fistula and wound finally healed.

This case illustrates a masked fistula which was first suspected about three months following lobectomy. It was definitely diagnosed when Chloresium was coughed up four and one-half months postoperatively. No further proof could be elicited until eleven months postoperatively when dye instillations showed that an abnormal communication existed. Bronchoscopies, other dye instillation and bronchograms before and after the positive test were negative.

CASE 4—L R, a 24 year-old Negro man, was admitted for treatment of pulmonary tuberculosis which originated in October, 1948. Bilateral productive disease with cavitation on the right was present. After numerous courses of streptomycin and PAS a three stage, seven rib thoracoplasty was done in October, November and December, 1950. A bronchogenic spread followed. Finally Jan. 29, 1952, a right upper lobectomy, resection of the superior segment, right lower lobe and decortication were done. Postoperatively his course was a relatively asymptomatic one but characterized by development of a pleurocutaneous sinus two and one-half weeks postoperatively. The sinus persisted and drained a moderate amount of serous material, but caused no other symptoms or signs. Methylene blue instilled into the sinus on Feb 23 1952, did not appear in the sputum. Bronchoscopies Feb 7, 1952, June 5, 1952, July 17, 1952, and Sept 18, 1952 showed no evidence of a bronchopleural fistula. The sinus tract appeared to be slowly healing and the patient had no complaints. As a matter of routine inquisitiveness on July 21 1952, Iodochlorol was instilled into the small sinus tract and, to our surprise, a nearly complete bronchogram of the opposite lung was obtained, thereby clearly demonstrating a bronchopleural fistula. Nothing further was done therapeutically, since healing seemed to be progressing slowly and spontaneously. In August,

bronchograms done in routine fashion failed to show the fistula. On Aug 31, 1952, the patient spontaneously coughed up a black silk suture, within ten days the sinus tract had completely closed and has stayed closed to date

The case clearly demonstrates persistent bronchopleural fistula which was absolutely asymptomatic and was discovered six months following resection by routine investigation of a persistent pleurocutaneous sinus tract by instillation of Iodoclorol

TABLE II EFFECTIVE INCIDENCE OR FREQUENCY OF VARIOUS DIAGNOSTIC SIGNS OF MEASURES EMPLOYED IN 24 CASES

SIGN *	POSITIVE	SUGGESTIVE
1 Pain, tightness, "bubbling" feeling	2	7
2 Fever	0	12
3 Cough with bloody sputum, worse on position change	3	5
4 Air on x ray (previously unseen or decreased)	0	10
5 Decrease in fluid level in chest as seen on x ray	6	0
6 Injection of sinus tract with radiopaque material	2	0
7 Injection or instillation of		
(a) Methylene blue	8	0
(b) Varidase	1	0
(c) Ether	2	0
(d) Tryptar	1	0
(e) Chloresum	1	0
8 Bronchograms	0	0
9 Bronchoscopy	8	0
10 Excess air (1) aspiration, (2) subcutaneous emphysema	7	3
11 Fistula found by later surgery	1	0

*Subjective 1-3

Objective 4-11

DISCUSSION

1 The highest number of fistulas were proved by methylene blue instillation into the suspected hemithorax. It is interesting to note, however, that in several cases of suspected fistulas, methylene blue instillations were repeated from 2 to 7 times before positive evidence of the dye could be found in expectorated sputum. Varidase, Tryptar, Chloresum, and ether had lesser application but occasionally pointed the way to a correct diagnosis

2 Bronchoscopic examination and needle aspiration of suspected pockets were the next most reliable diagnostic measures used to prove the existence of suspected bronchopleural fistulas. The likelihood of failure to visualize an existent fistula, however, is fairly great with bronchoscopy, contrary to the belief of many. In this series of 24 cases of fistulas, bronchoscopy was confirmatory in only 8 cases, or 33.3 per cent

3 The presence of seemingly limitless quantities of air, as obtained on aspiration, or seen in subcutaneous emphysema, etc., was also definitely diagnostic in 7 of the 24 cases, or 28.3 per cent, and highly suggestive in three instances

4 Coughing up bloody or pink-tinged fluid was considered diagnostic in three instances and highly suggestive in five other instances. When this could be correlated with x-ray visualization of a decreased fluid level and increased air pocket, it was considered positive, but in several instances it was impossible to do this because the pleural fluid drained through a pleurocutaneous sinus or thoracotomy tube

5 Decrease of fluid level on x-ray films was considered diagnostic also in six instances (not the same three mentioned in the preceding paragraph)

6 The subjective feeling of tightness, pain, or bubbling in the chest was diagnostic in two instances (where bubbling and feeling of blowing air occurred) and highly suggestive in seven other instances. It would appear that in the cases where pain or sensation of tightness is felt in the chest region several weeks or more following a pulmonary resection, the presence of a bronchopleural fistula and/or empyema pocket secondary to that fistula is extremely likely.

7 The injection of pleurocutaneous sinus tracts with a radiopaque material was conclusive in two instances, one of which was entirely unsuspected.

8 The presence of fever cannot be considered as more than a suggestive sign. This occurred in 12 instances, or 50 per cent of the 24 cases studied. Fever occurring after the first three postresection weeks should certainly direct efforts to proving or disproving, if possible, the existence of a bronchopleural fistula.

9 Bronchograms play a very limited role in proving the existence of a bronchopleural fistula even when one is almost certainly present. This is probably due to the high surface tension of the viscous oils used in bronchography. They tend to adhere to the margins of the small fistulas and form a protective film over the small orifice.

CONCLUSIONS

1 A definite clinical entity of so called masked or unrecognized bronchopleural fistula exists.

2 An attempt is made to define this entity and to state the factors involved in its recognition.

3 Even with the presumptive diagnosis made and with all the diagnostic armamentarium available, confirmation of these fistulas is often difficult.

4 Any or all of the applicable tests available should be employed and repeated as often as necessary. The initial failure of a "test" once does not mean it will subsequently be a failure.

5 It is suggested that any patent pleurocutaneous sinus be repeatedly investigated and considered as a possible omen of bronchopleurocutaneous communication.

6 It is suggested that a residual pleural space following any form of pulmonary resection be considered as potential evidence of an occult bronchopleural fistula and that every effort be made to prove its existence in order to institute early treatment and forestall serious complications.

EXPERIMENTAL TUBERCULOSIS

V EFFECTS OF LIGATION OF A PULMONARY ARTERY ON THE PULMONARY LESIONS OF INDUCED BOVINE TUBERCULOSIS IN RABBITS

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IN PREVIOUS studies^{1, 2} it has been shown that various experimental alterations of the pulmonary circulation in monkeys infected with human tubercle bacilli produce characteristic changes in the tuberculous process. "Arterialization" of the lung whether by anastomosis between a systemic and a pulmonary artery or by ligation of a pulmonary artery results in the development of massive conglomerate caseous lesions in the "arterialized" pulmonary tissue. Consistent changes of this type were found in a large series of monkeys irrespective of the size of the infecting dose of bacilli, the route of administration or the time of administration of the infecting dose in relation to operation. These observations were made in monkeys infected with human tubercle bacilli. In order to determine whether host species and bacterial strain variation would alter these observed phenomena, the present experiments were conducted. This report is concerned with the effects of "arterialization" of the lung, by ligation of a pulmonary artery, on the pulmonary lesions of induced bovine tuberculosis in rabbits.

METHODS

In these experiments 50 healthy adult rabbits weighing 2 to 3 kg were used. A standard dose of 1 cc of a suspension containing 10^{-4} mg of organisms per cubic centimeter of the same virulent *bovine* strain of tubercle bacilli was given to each rabbit intravenously. Five days later anesthesia was induced with intravenous sodium pentobarbital, 30 mg per kilogram of body weight. Because of the difficulty of oral intubation of the trachea in the rabbit, tracheotomy was performed in each case and a No. 12 Foley catheter inserted into the trachea. The catheter was connected to a mechanical insufflator through which passed 3 liters of oxygen per minute. All rabbits were then submitted to thoracotomy with full aseptic technique. In one group of animals the left pulmonary artery was isolated and ligated. In the remaining animals simple thoracotomy was done as a control. Great care was taken in each instance to obtain accurate re-expansion of the lung before closure of the thoracotomy wound. The catheter was then removed from the trachea and the small tracheal opening was closed with a single suture. The animals were kept in individual cages in the same isolation room and were fed stock diet until death.

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RESULTS

Of the 50 animals submitted to thoracotomy there were 18 anesthetic deaths and 14 other animals died within one to ten days after operation. Intolerance to anesthesia and postoperative atelectasis were largely responsible for the high mortality rate. Mechanical insufflation via tracheotomy was only partially successful in circumventing these problems. The remaining 18 animals (13 with pulmonary artery ligation and 5 with control thoracotomy) developed tuberculosis which progressed to a fatal termination in from 54 to 140 days. An autopsy was performed immediately after death in each case. Special care was taken to compare the character and extent of the tuberculous process in each lung and a detailed description was recorded. Both color and black and white photographs of the lungs were made as an aid in recording gross appearances. In addition the lungs from each rabbit were preserved in formalin for subsequent checking and comparison of the tuberculous lesions. Sections were taken for microscopic study and specimens were cultured for tubercle bacilli.

Results Following Ligation of Left Pulmonary Artery—There were 13 rabbits which survived operation and the immediate postoperative period. All had received the standard dose of bovine tubercle bacilli intravenously five days prior to surgery. These rabbits lived 54 to 114 days following inoculation. Pertinent data concerning these animals are summarized in Table I.

TABLE I. EFFECTS OF LIGATION OF PULMONARY ARTERY ON BOVINE TUBERCULOSIS IN RABBITS

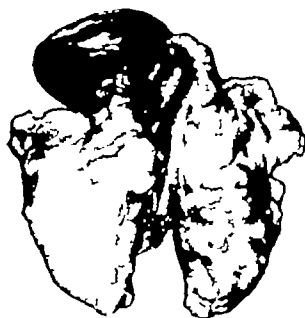
RABBIT NO AND PROCEDURE	SURVIVAL TIME AFTER INJECTION (DAYS)	DISTRIBUTION OF PULMONARY LESIONS AT AUTOPSY		
		EXTENSIVE CASEATION IN LUNG WITH ARTERY LIGATED	MORE EXTENSIVE IN OPPOSITE LUNG	NO DIFFERENCE IN LUNGS
Pulmonary Artery				
Ligated				
7 B	75	+++		
10 B	99	+		
11 B	101	++		
12 B	72	+		
14 B	79	-		
15-B	88	Marked atelectasis		
19 B	101	+++		-
27 B	83	Marked atelectasis		
28 B	54	+++		+
34-B	113	+		
40 B	85	-		
41 B	83	Some atelectasis		
48 B	71	+		+
Control Thoracotomy				
18 B	88			
42 B	75			+
45 B	116			+
49 B	99			+
50 B	140			+

In 10 of the 13 animals with interruption of the left pulmonary artery the extent of the pulmonary disease was greater in the left lung. Examples of the differences in the disease process in the two lungs are depicted in Fig 1. The left lung characteristically revealed large conglomerate caseous lesions in contrast with the right lung which presented smaller and less widely distributed

tubercles showing less conglomeration. Atelectasis of varying degree was present in the left lung in 8 of the 13 animals. In each of these 8 animals the caseous lesions in the left lung were less extensive than in the 5 animals whose lungs were entirely free of atelectasis. In 3 rabbits with a ligated left pulmonary artery there were no gross differences in the character or distribution of the lesions in the left or right lung. The left lung was atelectatic in each of these animals.



LEFT - DORSAL - RIGHT
RABBIT # 28B



LEFT - DORSAL - RIGHT
RABBIT # 11



LEFT - DORSAL - RIGHT
RABBIT # 19



RIGHT - VENTRAL - LEFT
RABBIT # 7B

Fig 1 — Autopsy specimens of lungs from rabbits submitted to ligation of left pulmonary artery following inoculation with bovine tubercle bacilli



LEFT - DORSAL - RIGHT
RABBIT # 18 B



RIGHT - VENTRAL - LEFT
RABBIT # 50 B



LEFT - DORSAL - RIGHT
RABBIT # 42-B

Fig. 2—Autopsy specimens of lungs from rabbits submitted to control thoracotomy following inoculation with bovine bacilli

Extrapulmonary lesions in these animals were often found in the liver, spleen, pericardium, and kidneys, but were much less extensive than those in the lungs. Microscopic study and bacterial cultures confirmed the presence of tubercle bacilli. Microscopic examination of the lesions in the two lungs failed to reveal any significant difference that was not apparent from gross examination.

Results Following Control Thoracotomy—Five rabbits of the group which was submitted to control thoracotomy survived operation and the immediate postoperative period. Each of these animals had received the standard dose of bovine tubercle bacilli intravenously five days before thoracotomy. These animals lived 75 to 140 days following inoculation. The autopsy findings are summarized in Table I. The lungs of these five rabbits revealed an equal distribution of small tubercles ranging from 2 to 6 mm in diameter. There was no difference in the character or extent of lesions in the two lungs. Typical findings are illustrated in Fig. 2.

COMMENT

The results of this investigation indicate that in rabbits with induced bovine tuberculosis, ligation of a pulmonary artery results in alteration of the disease in the affected lung. When the left pulmonary artery is ligated, the tuberculous process usually becomes much more extensive in the left lung than in the right. A lung thus deprived of a portion of its blood supply develops a more massive conglomerate caseous form of bovine tuberculosis than the contralateral lung of the same animal or both lungs of control animals.

These changes in the pulmonary lesions of rabbits infected with bovine tubercle bacilli appear to be identical with those observed in the "arterialized" lungs of monkeys infected with human tubercle bacilli.^{1,3} A fulminating tuberculous process resulting from arterialization of the lung thus seems to be a phenomenon which has no species or strain specificity.

Available evidence indicates that the more massive caseous tuberculous lesions which consistently developed experimentally in arterialized pulmonary tissue are the result of an enhanced growth of tubercle bacilli. Oxygen enhances the growth in vitro of both human and bovine strains of *Mycobacterium tuberculosis*,^{4,6} and, as has been pointed out previously,^{2,3} a lung or a segment of the lung with its pulmonary artery ligated is perfused only by fully oxygenated systemic blood via the bronchial arteries. Similarly, a lung which is arterialized by an anastomosis between the end of a systemic artery, such as the innominate, and the distal end of the pulmonary artery is perfused only by the fully oxygenated systemic blood from the anastomosis and that from the bronchial arteries. As has been shown previously,^{1,3} arterialization of the lung by either method results in severe fulminating tuberculosis in the affected lung. Increased oxygen tension in the arterialized pulmonary tissue with consequent enhanced growth of tubercle bacilli appears to be the fundamental factor responsible for the extensive pulmonary lesions.

SUMMARY

In rabbits with induced bovine tuberculosis, ligation of a pulmonary artery results in a more massive caseous form of disease in the affected lung. These

findings are identical with those previously observed in monkeys with induced human tuberculosis when submitted either to ligation of a pulmonary artery or to anastomosis between a systemic and a pulmonary artery. The present observations indicate that there is no species or strain specificity in this phenomenon which seems to be explained by an enhanced growth of tubercle bacilli as a result of increased oxygen tension in 'arterialized' pulmonary tissue.

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STAB WOUND OF THE HEART WITH DELAYED HEMOPERICARDIUM

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IN RECENT years there has been a decrease in the mortality of penetrating wounds of the heart. This has been brought about largely by increased awareness of the need for prompt diagnosis and treatment of acute cardiac tamponade. While the acute phase has been covered adequately in the literature, there is scant mention of the occurrence and management of delayed intrapericardial bleeding. This may be due to the rarity of the condition. The following case demonstrates the importance of keeping under observation patients who have been stabbed in the thorax, even though they may be clinically well for several days after injury.

REPORT OF CASE

A 39 year old Negro man was stabbed with a knife in the left hemithorax, June 21, 1953. He did not seek medical attention immediately, and during the next few days he was well except for some soreness in the chest. Because of this discomfort he saw a physician on June 27, six days after the accident. A roentgenogram of the chest (Fig 1) showed fluid in the left pleural space, but definite enlargement of the heart shadow could not be diagnosed. No definitive treatment was given at this time. On July 8, while doing heavy work, he became aware of precordial pain and exertional dyspnea, both of which persisted and led to hospitalization on July 13.

Examination disclosed a recent scar, 1 cm long, in the third left intercostal space adjacent to the sternum. There were numerous old scars on the chest and abdomen due to previous lacerations. His temperature was 100.2° F, pulse 100, and blood pressure 100/70 mm Hg. There was no venous engorgement, venous pressure was not measured. No cardiac apex pulsation was present and the heart was greatly enlarged to percussion. The sounds were of fairly good intensity. A pericardial friction rub was heard along the right sternal border. Hemoglobin was 12 Gm. A chest film (Fig 2), taken twenty two days after injury, showed tremendous enlargement of the cardiac shadow with the typical water bottle shape of pericardial effusion.

A diagnosis of hemopericardium due to stab wound of the heart was made. Pericardiocentesis by the paraxiphoid route were done on four occasions with the removal of 1,130 cc of dark nonclotting blood. A subsequent aspiration yielded less than 5 cc of blood. At this time the cardiac silhouette had receded but had not returned to normal (Fig 3). It was thought that any blood remaining in the pericardium was either clotted or loculated. Exploration was recommended to remove clots and repair any site of continued leakage. This was declined by the patient, and he left the hospital on July 25.

He returned to work and had no further symptoms. Follow up films have demonstrated gradual diminution in the size of the heart shadow. A roentgenogram on May 14, 1954 (Fig 4), eleven months after injury, showed a normal cardiac outline. An electrocardiogram made on May 14, 1954, revealed no abnormality.

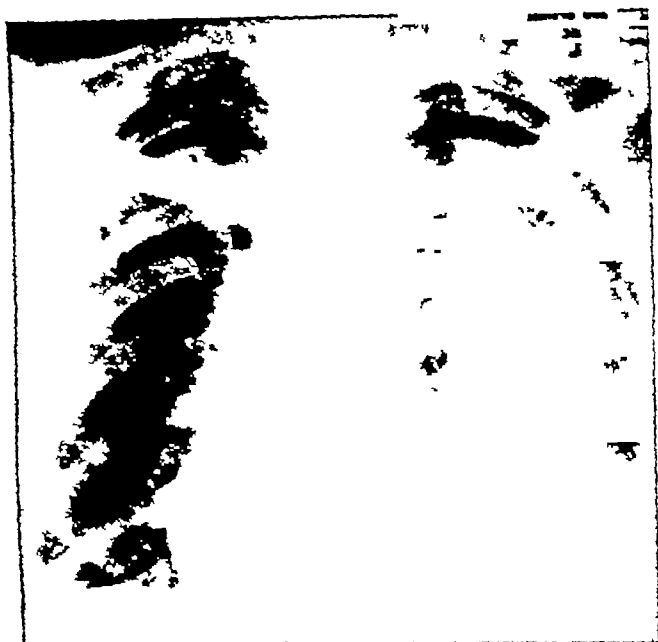


Fig 1—June 27 1953 six days after injury Fluid in left pleural space



Fig 2—July 14 1953 twenty two days after injury Massive hemopericardium.

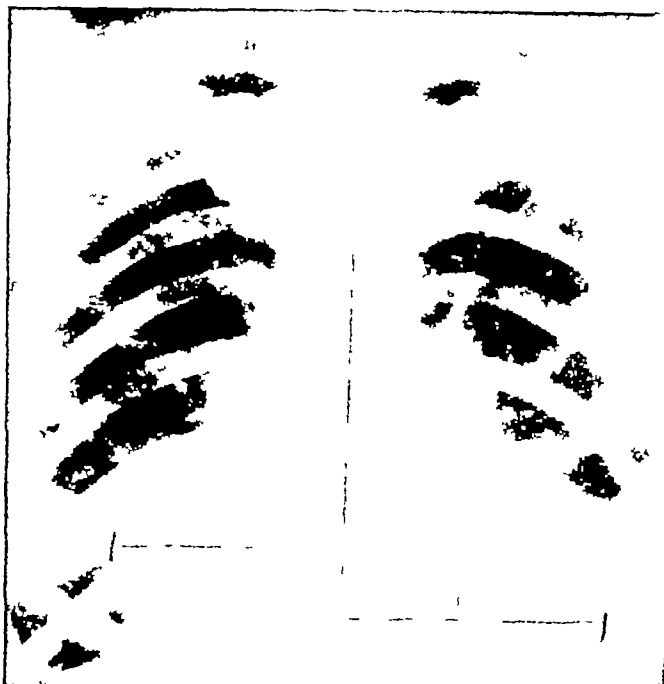


Fig 3—July 24 1953 Pericardial shadow smaller after aspiration of 1130 cc of blood
No more fluid could be aspirated

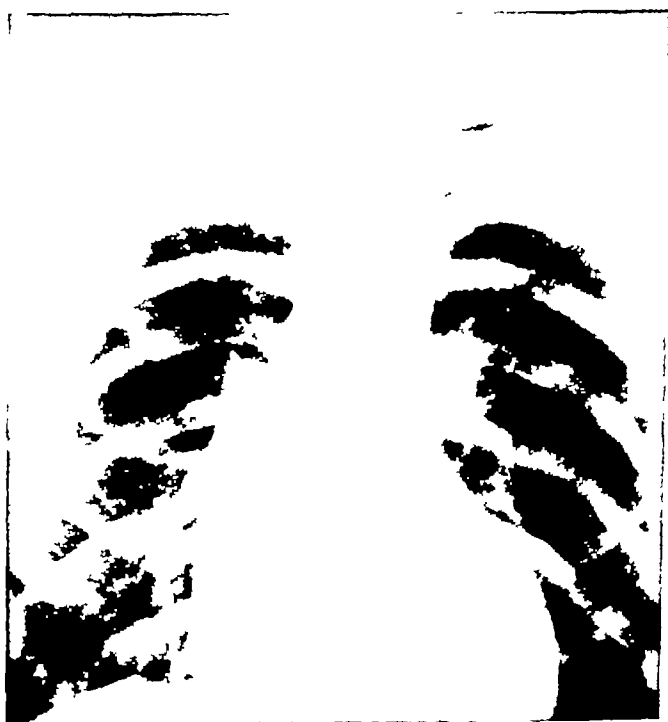


Fig 4—May 14 1954 Normal cardiac silhouette eleven months after injury

COMMENT

Six days after a stab wound of the chest a roentgenogram showed fluid in the left pleural space but did not suggest intrapericardial bleeding. Symptoms gradually appeared and a film taken twenty-two days after the injury showed a tremendous pericardial shadow. With this sequence of events, it is likely that the wound of the heart bled initially the blood escaping from the pericardium to the pleural space. The myocardial and pericardial wounds then became sealed. Later the heart wound bled again but this time the blood remained in the pericardial sac because the pericardial wound had healed. The secondary bleeding was probably slow as the pericardium had time to stretch and tamponade did not occur.

Since operation was not done the exact site of cardiac injury is not known. It is known however that wounds of the ventricle are more likely to heal spontaneously than are wounds of the atrium. In a report of a single case and a review of eleven others¹ in which hemopericardium became evident more than forty-eight hours after injury, the wound was found to involve the ventricle in each instance. For these reasons it is probable that the wound in the present case involved the ventricle.

After the last pericardiocentesis, when no more blood could be aspirated and the heart shadow had not returned to normal enzymatic débridement was considered. This was not done because of the possibility of further hemorrhage from liquefying or dislodging an unstable clot.

The normal electrocardiogram eleven months following injury is not surprising since there is a tendency for the tracing to return to normal with the passage of time after penetrating wounds of the heart.²

SUMMARY

A case of hemopericardium appearing some time between six and twenty-two days after a stab wound of the heart is presented. Delayed hemorrhage from a heart wound is rare and emphasizes the importance of keeping these patients under observation.

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EOSINOPHILIC GRANULOMA OF RIBS

REVIEW OF LITERATURE AND REPORT OF TWO CASES WITH FOUR AND SIX AND ONE-HALF YEAR FOLLOW-UP, RESPECTIVELY

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EOSINOPHILIC granuloma of bone is a benign, chronic, progressive, destructive process which may mimic any neoplastic or inflammatory bone lesion. Most authorities consider eosinophilic granuloma of bone as a monosymptomatic form of Letterer-Siwe's or Hand-Schüller-Christian's syndromes, and the relationship between these disturbances and eosinophilic granuloma of bone has been discussed at length by Otani and Ehlich,⁷⁰ Mallory,⁶⁸ Green and Fairber,⁴¹ Engelbreth-Holm, Teilum, and Christensen,²⁷ and by Thannhauser.⁸⁰ A working classification summarizing these disease relationships has been outlined by Weinstein, Francis, and Sprockin¹⁰⁴ from Mallory's suggestions, and is presented in the first three paragraphs of Table I. Henderson, Dahlin, and Bickel⁴⁹ do not feel that eosinophilic granuloma of bone is an early phase in the life cycle of Hand-Schüller-Christian's syndrome. They feel that the term eosinophilic granuloma should be used to describe bony lesions in patients only in the absence of visceral involvement and provided that not more than two bony lesions are present. Since publication of Mallory's suggestions in 1942, many patients have been reported in whom eosinophilic granuloma of bone has been associated with various similar lesions in soft tissues, and yet these patients have not shown signs suggesting Letterer-Siwe's or Hand-Schüller-Christian's syndromes. The first such patient was reported by Arnold³ in 1946, when he described the occurrence, in a 37-year-old white woman, of an eosinophilic granuloma of the clavicle associated with scattered, bilateral infiltrations of the lungs, which were assumed to be similar to the granuloma in the bone. The lesion in the clavicle healed spontaneously, and the lesions in the lungs cleared following x-ray therapy. In 1947, Weinstein and associates¹⁰⁴ described a patient who had multiple eosinophilic granulomas of bone associated with pulmonary infiltrations. Similar reports have been made since by Zinneman,¹⁰⁶ Dickson,²⁴ Kruger, Priekman, and Pugh,⁵⁰ and by Walthard and Zuppinger.¹⁰¹ Patients demonstrating combinations of eosinophilic granuloma of bone combined with skin lesions have been reported by Curtis and Cawley²³ and by Kendrick and Woodruff.⁵⁶ A patient reported by Schnoff⁸⁸ presented an eosinophilic granuloma of bone associated with an anal fistula containing similar tissue. Because of this last group of patients, we have taken the liberty of adding to Mallory's observations the suggestions in the fourth paragraph of Table I.

The subsequent discussion is confined to eosinophilic granuloma limited to the skeleton, and without visceral lesions, except in a few instances where soft

TABLE I

<i>Letterer Siwe's Syndrome</i> (reticuloendotheliosis, aleukemic reticulosis, nonlipid histiocytosis)	
Usually children under 2 years of age, fever, skin rash, anemia, purpura, splenomegaly	
Histologically Proliferation of monocytes and clasmatoocytes in bone, skin, lymph nodes, and spleen, with or without deposits of lipids	
<i>Hand Schüller Christian's Syndrome</i>	
Usually older children, defects in cranial bones, exophthalmos, diabetes insipidus	
Xanthoma deposits with or without eosinophils	
Lymph nodes may show typical eosinophilic granuloma with or without deposits of cholesterol	
<i>Eosinophilic Granuloma of Bone</i>	
Children or adults	
Single or multiple lesions of bone	
Good general health, low grade fever, leukocytosis with eosinophils, local tumor some times painful	
Cured by local excision, x ray, or occasionally spontaneously	
<i>Eosinophilic Granuloma of Bone and Soft Tissue</i>	
Usually adults	
Bony lesion(s) with granulomas of skin or lungs	
Some have diabetes insipidus	
Chronic course, soft tissue granulomas tend to heal bone lesions tend to recur at old or new sites	

The first three paragraphs were tabulated from Weinstein Francis and Sprocklin²⁴ from Mallory's suggestions. The fourth paragraph has been added since that time on the basis of case reports published subsequent to 1942.

tissues adjacent to a granuloma of bone were involved by direct extension of the lesion in the bone. These criteria are not inflexible, however, and will vary with the thoroughness and duration of the clinical study and follow-up period.

Eosinophilic granuloma of bone was probably first described* by Finzi,³² in 1929, in reporting a myeloma with prevalence of eosinophilic cells occurring in the frontal bone of a 15-year-old boy. In 1930, Mignon³³ described a granulomatous tumor occurring in the frontal bone of a 17-year-old boy. Fraser,³⁴ in 1935, described lipoid granulomas occurring in two infants, one patient having a single lesion in the ilium and the other having multiple bony lesions, each running a benign course and apparently healing after the use of radium packs. Whether these were eosinophilic granulomas healing by lipidization and fibrosis is not certain. In 1938, Schairer⁵ described two instances of osteomyelitis with eosinophilic reaction in the parietal bones of 9- and 10-year-old children. In 1940, Otani and Ehrlich⁶ described seven patients with granulomas of bone simulating primary neoplasms, and Lichtenstein and Jaffe⁶⁴ reported three cases with similar lesions and first suggested the term "eosinophilic granuloma of bone."

To date about 187 patients with eosinophilic granulomas of bone have been reported in the literature, and herewith are reported two more patients demonstrating solitary eosinophilic granulomas of the ribs, making the total 189.

The process is a single or multiple one, beginning in the medullary areas and tending to spread outward with erosion and perforation of the cortex, and sometimes extending into the surrounding soft tissues. There has been no particular pattern of distribution shown in the bones involved, and there does

*Mr. Thomas Smith³⁵ in 1865 described three large defects in the skull of a 4-year-old child which had previously suffered from impetigo of the scalp and had died of pertussis. Smith described the openings as congenital defects of the bone and no histologic data is presented.

not seem to be any predilection for any portion of a given bone. Lesions have been described in all bones excluding the patella, ulna, tarsals, carpals, and phalanges of the toes. The bone is replaced by soft, yellowish or brownish tissue, the color probably resulting from areas of necrosis and hemorrhage. The microscopic appearance of the tissue may vary according to the stage of the lesion. In the first stages there are sheets of eosinophils and histiocytes with some areas of giant cells, eosinophilic myelocytes, neutrophils, lymphocytes, and plasma cells. Cellular products of destruction and repair may be present with lipophages and other phagocytes. Otani and Ehrlich³⁶ and Green and Farber⁴¹ feel that, in the late stages of the disease, eosinophils tend to disappear and there is an increase in fibrosis and large mononuclears, and also, in the healing stages, large mononuclears may undergo vacuolization and appear as lipophages. Still later in the process, granulation tissue is replaced by connective tissue, which may be converted into bone.

The etiology of eosinophilic granuloma of bone is unknown. It is felt to be inflammatory, but no viral or bacterial cause has as yet been demonstrated. Preceding trauma has been elicited in the histories of some patients, but it is probably more coincidental than causative. A functional disorder of the suprarenal cortex has been suggested by Frischknecht,³⁶ because he observed in a child with eosinophilic granuloma of bone, familial hirsutism, increased sodium and cholesterol in the blood, abnormal sugar retention, decrease in circulating eosinophils, and advanced ossification of carpal bones.

Of 189 patients so far reported, 119 were males, 48 were females, and the sex of 22 patients is not listed. A distorted sex variation might occur in the literature due to reports of many male patients from the armed forces. Yet, in a series of 10 children reported by Green and Farber⁴¹ in 1942, there were 9 males and 1 female. The age distribution, the relative frequency of single and multiple lesions, and the occurrence in various bones are given in Table II.

Local symptoms and signs will vary widely depending on the location of the lesion, whether single or multiple, and the degree of direct invasion of surrounding tissue. Multiple lesions may be present and yet only one may be giving rise to symptoms. There may be local pain and tenderness with some swelling and even fluctuation suggesting abscess. Involvement of skull bones may cause various disturbances of the cerebral cortex or extension of the disease into the middle ear,⁹² mastoid process,^{10, 47, 101} accessory nasal sinuses,¹⁰ or orbit.^{13, 48} Lesions in the maxilla or mandible may cause destruction of tooth buds or loosening of teeth already erupted with a syndrome suggesting chronic gingivitis.^{92, 100} Vertebral involvement may, by direct extension, result in spinal cord disturbances.⁸¹ Granulomas in the long or flat bones of the extremities, chest, pelvic or shoulder girdle may result in limitation of motion or pathologic fractures.^{33, 79}

Systemic disturbances are usually mild and may include malaise, slight weight loss, slight fever, and occasionally leukocytosis. Anemia has been reported in one child, probably the result of x-ray therapy.⁴¹ Eosinophilia, varying from 4 to 12 per cent, has been described by many authors although Frischknecht³⁶ cites one instance of a decrease in circulating eosinophils. Lichtenstein

TABLE II AGE AND SEX DISTRIBUTION, RELATIVE FREQUENCY OF SINGLE AND MULTIPLE LESIONS, AND OCCURRENCE IN VARIOUS BONES OF LESIONS OF EOSINOPHILIC GRANULOMA IN 189 PATIENTS

Skull	102	Single	132
Ribs	54	Multiple	42
Femur	31	Not Stated	15
Pelvis	24		
Mandible	18		
Humerus	16	Not yet reported in tarsals, carpals, patella, ulna or phalanges of toes	
Vertebra	14		
Scapula	12		
Clavicle	8		
Tibia	7		
Radius	3		
Fibula	2	Males	119
Sternum	2	Females	48
Metacarpal	1	Not stated	22
Metatarsal	1		
Phalanges of hand	1		
	Under 10 years	62	
	10-19	44	
	20-29	38	
	30-39	17	
	40-49	8	
	50-59	3	
	Not stated	17	

and Jaffe⁶⁴ describe bone marrow biopsy in one patient showing an increase in eosinophilic myelocytes and young eosinophilic granulocytes to comprise almost 15 per cent of the total marrow cells. Kipp and Fisher⁶⁵ describe the presence of large multinucleated histiocytes in the sternal bone marrow of a 22-year-old woman who had an eosinophilic granuloma of a rib, the large cells in the sternal marrow being structurally identical with cells seen in the rib marrow adjacent to the granuloma. All other studies on body fluid constituents such as calcium, phosphorus, cholesterol, cholesterol esters, blood lipids, urinalyses, and phosphatase have been described as within normal limits.

The x-ray appearance of eosinophilic granuloma is that of a small or large, irregularly destructive, radiolucent zone, with or without periosteal reaction, bone consolidation, or pathologic fracture. In flat bones the lesion is usually circumscribed, sharply delineated, or punched out. In the ribs there may be destruction with expansion of the cortex, perforation, pathologic fracture, periosteal new bone formation, and thickening of the overlying tissues. The differential diagnosis includes all the neoplastic and inflammatory diseases of bone.

The course of untreated lesions varies greatly. They may progress at various rates, stop at any stage, remain stationary, or recede and heal slowly and spontaneously by fibrosis and reossification, leaving no trace in subsequent x-ray examinations. Some do not heal, and even though single or multiple granulomas have been observed over periods of years to remain stationary,⁴⁰ they apparently do not harm the patient's general health save by local invasion of surrounding tissue or pathologic fractures.

Accurate diagnosis cannot be made without biopsy, and a single lesion may be biopsied or excised, depending on its location. If the lesions are multiple, biopsy or excision of one or more will establish the diagnosis, and it may reasonably be assumed that the other lesions are identical.

Recommended methods of treatment consist of curettage or excision, with or without x-ray therapy. Brody and Gillespie¹⁵ describe the use of adrenal corticotropic hormone in a 24-year-old man with eosinophilic granulomas in the mandible, femur, pelvis, and lumbar vertebrae. Use of the drug was followed by fever and an increase in pain, it was stopped after 600 mg had been given. X-ray therapy was then used and did aid in alleviating the patient's pain. This patient also had transitory infiltrations of his lung fields. Henderson and Thompson¹⁶ described the use of adrenal corticotropic hormone in a 32-year-old man with an eosinophilic granuloma of the mandible associated with pulmonary infiltrations. Fifteen milligrams of the drug was given every six hours for four days. No undesirable effects occurred, but no benefit resulted, and no change was noted in the appearance of the lung lesions by roentgen examinations. X-ray therapy to the mandible over a period of four months was followed by healing of the lesion in the bone. No x-ray therapy was given to the lungs, and the pulmonary lesions remained unchanged, although the patient felt entirely well at the time of the report.

Biopsy or curettage may or may not be followed by healing. Some have not healed after curettage, and yet other undisturbed and untreated lesions in the same patient have healed spontaneously.¹⁰⁴ Some granulomas of the skull, unhealed after curettage, have required subsequent insertion of prosthetic skull plates. Ponseti⁹ describes nonhealing of a granuloma of the femur in a 46-year-old man one year following curettage and insertion of a bone graft. X-ray therapy was then given, after one month, during which the patient had sustained a pathologic fracture at the site, a large defect was still present, and the leg was amputated. The patient was described as well five years after amputation. Michael and Norcross¹² described improvement in a 21-year-old man following laminectomy and x-ray therapy for eosinophilic granuloma involving the ninth and tenth thoracic vertebrae, the lesion having involved the spinal cord with resulting weakness of the legs and sensory loss up to the level of the tenth thoracic segment. It would appear that surgery should probably be limited to that necessary for biopsy, to correct deformities such as pathologic fractures or to relieve damage done by direct invasion of surrounding tissues.

Most authorities feel that x-ray therapy should be used, although there is little uniformity of opinion regarding its efficacy or the technical aspects of its use. Response to irradiation varies greatly. Some lesions have shown an initial increase in size while being irradiated and have then slowly healed. Some patients have developed new lesions while the original ones were being irradiated. In Green and Farber's⁴¹ group of patients treated with roentgen irradiation, all were reported as healed within nineteen months. Cooley and Carlson¹⁰ report one instance of a healing period of three and one-half years following x-ray therapy.

Therapeutic results following small infrequent doses of irradiation seem to be about the same as those following larger, more frequent exposures. Dundon, Williams, and Laipply²⁶ report variations in total dosage from 400 to 500 r (all) to 1200 to 1500 r (all), usually given in divided doses of 200 to 300 r

daily or even monthly, and these regimes have been followed by complete healing of skull lesions in from five to nineteen months. Hamilton and his associates¹⁶ treated six patients by combined surgical (biopsy or curettage) and roentgen therapy, using a 220 kv machine and the following factors: 220 kv 15 Ma, 0.5 to 1.0 mm Cu plus 1.0 mm Al filter, HVL 0.9 to 1.35 Cu 50 cm TSD. Dosage varied from 150 r every other day to three times a week or 200 r every day or every other day and the portals were of adequate size to cover individual lesions or involved areas. They felt that x-ray therapy helped to relieve pain and that it decreased limitation of function in an extremity where function had previously been limited but that subsequent x-ray study did not show a greater or earlier degree of bone repair in irradiated areas as compared to non-irradiated areas. Green and Farber¹¹ describe two types of irradiation with similar results from each: (1) 160 kv 5 Ma 40 cm distance 4.0 mm Al filter giving 400 r for two doses on successive days or successive months; (2) 160 kv 5 Ma 40 cm distance 0.25 mm Cu and 1.0 mm Al filter giving a total dosage of 1500 r over a six-day period.

Many patients have been reported who have eosinophilic granuloma of bone associated with various pulmonary infiltrations.^{3, 24, 40, 50, 77, 80} A much larger number of patients has been described where pulmonary eosinophilic infiltrations have sometimes been associated with eosinophilia in the peripheral blood but in the absence of bone lesions.^{28, 30, 77, 103} The relationship between these two groups of patients is unknown. In Brody and Gillespie's patient¹⁵ mentioned previously suffering from multiple eosinophilic granulomas of bone with pulmonary infiltrations the bone pain was apparently worse following administration of adrenal corticotrophic hormone and no change occurred in the lung lesions. The patient of Henderson and Thompson⁵⁰ derived no apparent benefit from adrenal corticotrophic hormone although the symptoms were not aggravated by the drug. Vines and Clark²⁸ have recently discussed the treatment of a woman suffering from the eosinophilic pneumonopathy syndrome without any bone lesions where the use of adrenal corticotrophic hormone was apparently lifesaving. The significance of the different reactions of these patients to this drug is unknown. Discussions of the various classifications of pulmonary eosinophilia have been presented by Apler and Grant² and by Crofton and his associates.²²

CASE REPORTS

CASE 1—E. L., a white man, aged 44 years, was admitted to the Nazareth Hospital, Jan. 5, 1948, with a history of a painful swelling over the right tenth rib in the midscapular line beginning eight weeks prior to admission. There was no history of injury. X-ray examination of the ribs done elsewhere, at this time was reported as negative. The swelling and pain subsided in a few days but some tenderness persisted. Six weeks prior to admission, when turning over in bed he experienced a sudden severe pain in this area. The pain decreased in severity in the following few days but some discomfort persisted for three weeks. Repeat x-ray examination of the ribs on Dec. 16, 1947, showed an area of rarefaction in the posterior portion of the right tenth rib, with a pathologic fracture present (Fig. 1). At the time of admission, physical examination was completely negative save for moderate tenderness on pressure over the involved portion of rib. The blood count was normal and there were no eosinophils seen in the blood smear. Urinalysis was negative save for a 2 plus Sulkowitch test indicating 8 to 11 mg of calcium per 100 c.c. of urine. The blood calcium



Fig 1—Case 1 Roentgenogram of ribs made Dec 16 1950 showing destruction of bone in right tenth rib



Fig 2—Case 1 Microscopic appearance of lesion

was 13.4 mg per cent and the blood phosphorus was 2.4 mg per cent. These studies were repeated and the values were 9.8 mg per cent and 2.8 mg per cent for calcium and phosphorus, respectively. Under local anesthesia with Novocain, a 2 inch segment of this rib was resected subperiosteally. The patient's postoperative course was uneventful. The report of the microscopic examination of the tissue by Dr. Emmett Ciccone was that it was compatible with eosinophilic granuloma of bone (Fig. 2).

Fig 3



Fig 4

Fig 3—Case 2 X ray appearance of destructive lesion of left fifth rib. Film made March 27 1950. Compare with Fig 4 which shows appearance one month later.
 Fig 4—Case 2 X ray appearance of destructive lesion of left fifth rib. Film made April 26 1950.

In August, 1948, the patient had symptoms and signs of a herniated lumbar intervertebral disc. X ray examination of the spine showed only narrowing of the fifth lumbar interspace. X ray examination of the lung fields was negative. In January, 1950, repeat X ray examination of his lung fields was negative. In June, 1954, X ray examination of the

ribs was entirely normal save for the surgically produced defect. Lung fields at this time, as well as a complete radiologic bone survey done by Dr. John Fetter, were completely negative. This patient's general health is excellent six and one half years after the resection of an eosinophilic granuloma of a rib.

CASE 2—M. McT., a white woman, aged 53 years, was admitted to the Nazareth Hospital on April 26, 1950, with a history of pain over the left, upper posterior chest of six weeks' duration. There was no history of trauma and the patient had no other symptoms. X-ray examination of the ribs made on March 27, 1950, showed a destructive lesion in the posterior portion of the left fifth rib (Fig. 3). Repeat x-ray examination at the time of admission showed that the lesion had progressed (Fig. 4). X-ray studies of the kidneys and the entire gastrointestinal tract were negative. A small cervical polyp was excised and histologic examination of this showed it to be a mucocoele of a pedunculated Nabothian cyst. Blood counts on three occasions were normal with 1 to 3 per cent eosinophilia. The urine gave a negative Sulkowitch test, and was negative for Bence Jones protein on two occasions. The total serum protein was 9.6 Gm. per cent, with albumin 6.3 Gm. per cent and globulin 3.3 Gm. per cent. The serum chlorides were 580 mg. per cent and the calcium was 11.5 mg. per cent. The basal metabolic rate was +3, and blood sedimentation rate was 19 mm. in 60 minutes.

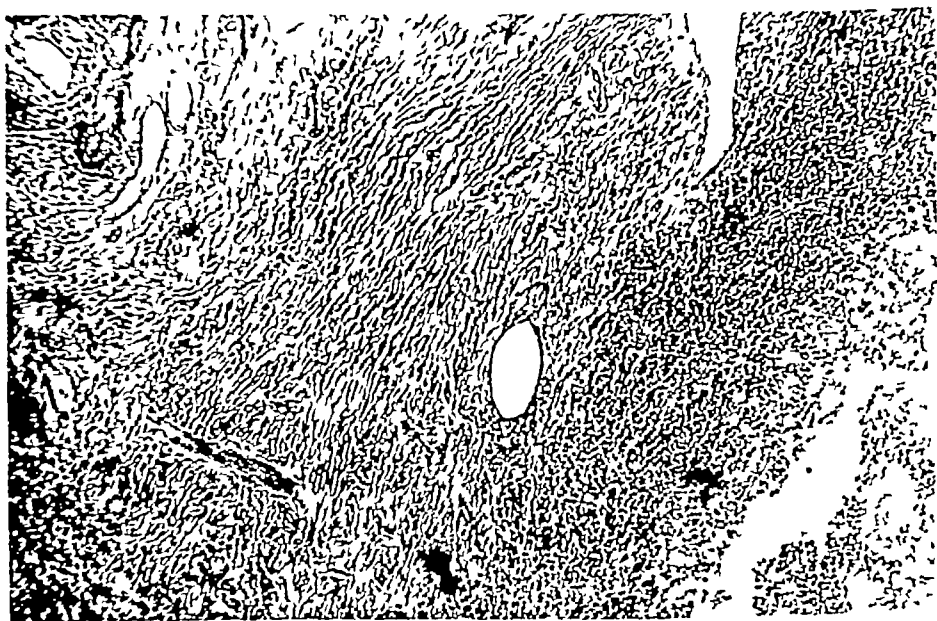


Fig. 5—Case 2. Microscopic appearance of lesion.

On May 3, 1950, the involved segment of rib was removed en bloc together with the attached intercostal muscles and parietal pleura. The patient's postoperative course was uneventful. Histologic examination of the specimen by Dr. Emmett Ciccone showed eosinophilic granuloma of bone (Fig. 5).

Aspiration biopsy of sternal marrow done six months after operation revealed normal marrow. Complete radiologic bone survey done in June, 1954, by Dr. John Fetter was normal save for the surgically produced rib defect. X-ray examination of the lung fields at this time was negative. This patient's general health four years after the resection of the eosinophilic granuloma of the rib, is normal in every way.

SUMMARY AND CONCLUSIONS

1 Eosinophilic granuloma of bone, although possibly a form of Letterer-Siwe's and Hand-Schüller-Christian's syndromes and possibly related to syndromes where bone and skin, or bones and lungs are involved, can occur in one or more sites limited to the skeleton and can run a chronic benign course with complete healing. One hundred and eighty-nine such patients have so far been reported.

2 The finding of a single eosinophilic granuloma in one bone should initiate a complete skeletal survey, as well as a careful search for any visceral involvement with particular attention to skin, lymph nodes, bone marrow, lungs, liver, and spleen or any evidence of diabetes insipidus, exophthalmos, or mental retardation.

3 In all patients where lesions are limited to the skeleton healing will probably occur, the outlook improving with the age of the patient. In infants and children the prognosis must always be guarded in view of the possible development months to years later, of Letterer-Siwe's or Hand-Schüller-Christian's syndromes. In adults, the prognosis, although better than in children, should be guarded against slow or nonhealing subsequent recurrences, new lesions, or involvement of lungs or skin.

4 Eosinophilic granuloma of bone is diagnosed by biopsy only. Surgical treatment is probably best limited to biopsy or treatment of sequelae. X-ray treatment of lesions is apparently of benefit in relieving pain and may aid in repair. Its use seems indicated until a better method becomes available.

5 Two patients are reported each having a solitary eosinophilic granuloma of a rib. Both patients are now entirely well, four, and six and one-half years respectively after resection of the involved rib segments.

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A MANEUVER FOR EXTRACTING CLOTS FROM THE ATRIUM AT THE TIME OF MITRAL COMMISSUROTOMY

WITH REPORTS OF FOUR CONSECUTIVE CASES

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THE surgical treatment of mitral stenosis by commissurotomy has become well established in the last few years. Over 1,000 operations have been performed at this Clinic alone.

The usual technique of performing the commissurotomy is to enter the left atrium with the right index finger through an incised area in the left auricular appendage that has been previously enclosed by a purse-string suture. The commissurotomy may be performed either with the guillotine knife or merely by digital pressure, as the case may be.

One of the most dreaded complications is that of finding a ball thrombus in the auricle attached by a very thin pedicle and waving freely in the blood stream. Very often these clots are 3 to 4 cm. in diameter and the pedicle gives a sensation of thin tentacles of seaweed with the ball clot attached to the end of the tentacles.

During any manipulation of the left auricular appendage, the left common carotid artery and the right innominate artery are clamped as previously described.¹ While this technique has reduced the hazard from clots as far as emboli to the brain at the time of surgery are concerned, the threat is still present after surgery if the clot is not removed. In addition, if the clot breaks loose at the time of surgery, the embolus may go to a visceral artery, where it may be impossible to remove it, or, if the embolus is large enough, it may completely block the aortic orifice, causing acute coronary insufficiency and ventricular fibrillation.

We wish to describe a maneuver that one of us has used to remove clots successfully in four consecutive cases during the past six months (W. L. J.).

Clots in the left auricular appendage are suspected if the surface of the appendage is fibrotic or the size is contracted. Palpation of the appendage is carried out with the left common carotid and right innominate arteries constricted.

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If the left auricular appendage appears clotted or a clot is palpated in the left atrium, the operator's left index finger is ungloved by stretching the glove from the finger tip and amputating the end. The use of the ungloved finger is a very important part of the technique, as much greater traction may be exerted. We have been able to control any postoperative infection by use of massive doses of antibiotics. At the time a clotted atrium is found, the risk of infection seems slight compared with the risk from the breaking loose of a large embolus. We therefore feel that use of the ungloved finger is justified. After the clot is gently palpated, it is compressed between the index finger and the atrial wall (Fig 1, a). It is then manipulated along the lateral wall of the atrium as far out of the appendage as can be attained without losing it (Fig 1, b).

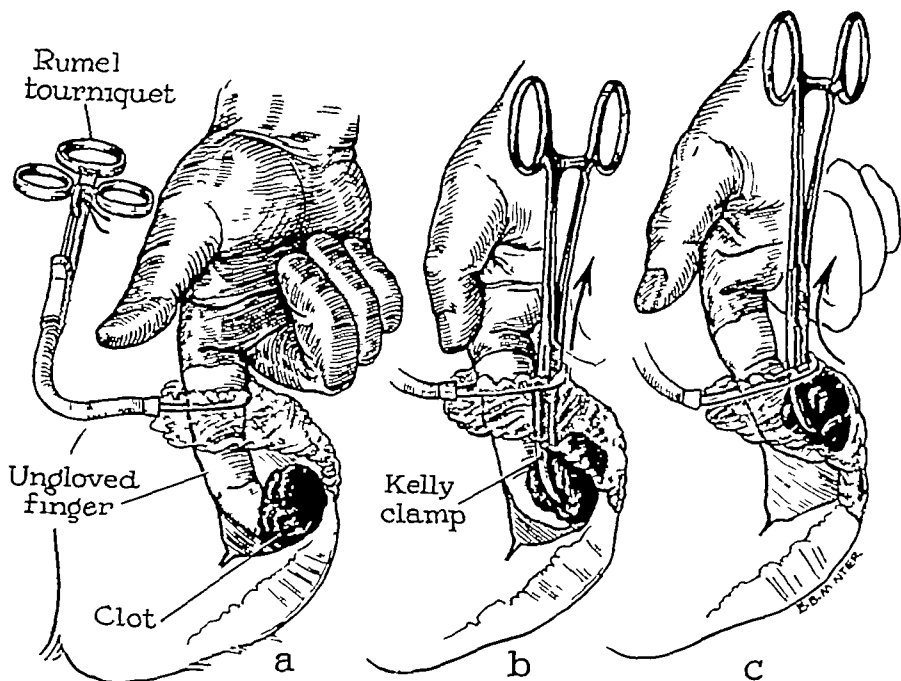


Fig 1—*a* The clot is gently compressed between the ungloved finger and the left atrial wall. *b* Traction is exerted on the clot, and it is manipulated into the left auricular appendage. A curved Kelly clamp is then inserted between the finger and the appendage. *c*, The clot is grasped with the curved Kelly clamp by sense of position.

The assistant holding the Rumel tourniquet is told to loosen the tourniquet to its full extent and under no circumstances to tighten it until the order is given. In some cases, it may be necessary to tell the assistant to put the tourniquet down, as the reflex to tighten it when the second stage of the maneuver is carried out may be very strong. It is vital when the next step is performed that the assistant does not tighten the tourniquet.

Whole blood is started at this time and the anesthesiologist is asked to have a pressure apparatus available for pumping large quantities in quickly.

A curved Kelly clamp is then inserted between the finger and the appendage. The clot is grasped with the Kelly clamp by a sense of feel the curved outline of the clamp usually following the wall of the appendage (Fig 1 b)

With a quick motion the finger, clot, and clamp are then withdrawn simultaneously and no effort is made to clamp the appendage. A large gush of blood ensues (Fig 2, b). The hand at this time is in supination due to the natural maneuver of extracting the finger and the clot from the appendage. In the second between supination and pronation that is almost reflexly carried out when the eye sees the gush of blood, it has been our experience that any loose or unattached clots will wash out (Fig 2 b). Pronation and grasping the appendage between the thumb and forefinger are then carried out. No effort to control the bleeding by the tourniquet is used as the time necessary to give the two seconds of bleeding after the finger is withdrawn can be attained only by the operator. Controlling a bleeding auricle by pressure between the finger and thumb is a simple, safe, sure procedure, first demonstrated at this Clinic by M T Lacy a resident at the time.

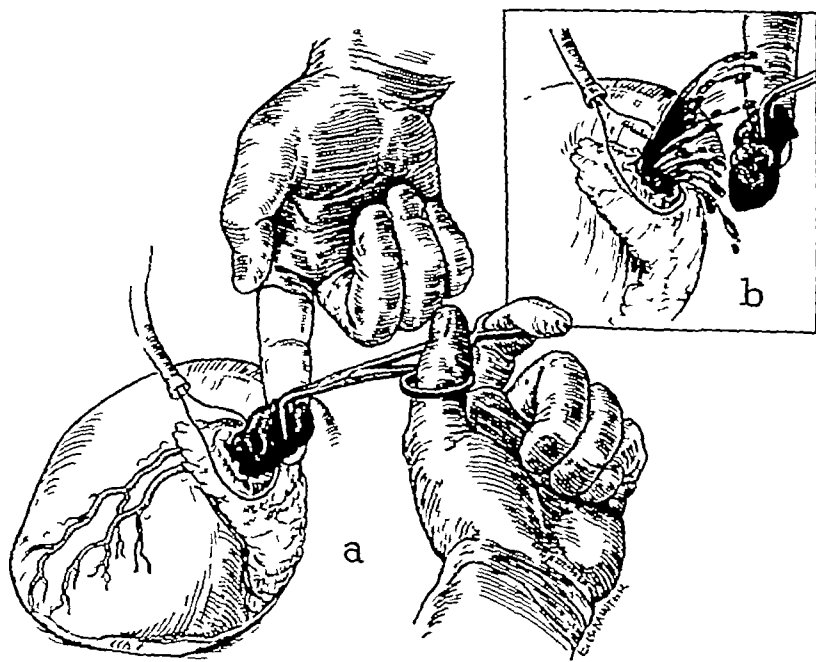


Fig 2—*a* Finger, clot, and Kelly clamp are withdrawn simultaneously. No effort is made to clamp the appendage. The Rumel tourniquet is loose. *b* Unattached clots wash out in the second before the appendage is grasped by the operator.

It may be stated that the ungloved finger is used more or less routinely at this Clinic and, therefore, is always able to meet the emergency of finding an unexpected ball thrombus in the auricle.

Four case histories in which this maneuver has been used are briefly presented to illustrate the different types and sizes of clots that have been removed.

CASE REPORTS

CASE 1—Mrs T, a 40 year old white woman, was referred for possible cardiac surgery on Jan 7, 1954. History revealed that she was reared in Europe until she was 9 years old, and she had no memory of ever having had rheumatic fever. Between the ages of 2 and 9, she had severe whooping cough, lasting seven months. Her heart disease was detected twenty two years ago, when she was pregnant with her only child, in the course of a routine checkup. She was very sick all through the pregnancy and was under medical supervision throughout. She was delivered at home. Her family physician warned her against having any more children.

She had often felt tired in the past one and one half years and had been aware of irregular heart action for the past three years. For three months before admission her shortness of breath had become more acute and she had developed paroxysmal nocturnal dyspnea and orthopnea. Digitalis was prescribed at this time.

One week prior to admission she had a left hemiplegia, with a very brief period of weakness of the left arm and the left leg, associated with loss of speech. She recovered the function of speech and the use of the left arm and leg within about an hour.

Past medical history revealed that, six years before, she had developed menorrhagia followed by episodes of weakness. She consulted a physician who made a diagnosis of fibrotic tumor of the uterus. Radium was instilled in 1949. X ray treatments were administered six months later. In spite of this treatment, menorrhagia and weakness persisted. For this reason, in 1951 abdominal section was performed and the fibroid uterus was removed. This brought about the condition of surgical menopause.

Physical examination revealed a tall, thin woman of 40 years, with dyspnea on exertion. Pertinent findings of the chest examination were as follows: chest was asymmetrical with kyphoscoliosis, examination of the lungs revealed normal breath sounds and a few moist sounds, examination of the heart revealed the rhythm to be that of auricular fibrillation. Blood pressure at the time of admission was 120/86, over the apex, a midlate diastolic murmur could be heard, pulmonic second sound was accentuated, no other thrills or murmurs were detected, and the liver was one finger below the costal margin.

Electrocardiogram showed auricular fibrillation with a well controlled ventricular rate and indicated clockwise rotation of the heart.

Fluoroscopy revealed diffuse dilatation of the heart. The cardiac contractions appeared to be diminished, and the silhouette reminded one of an enlarging pericardial effusion. The pulmonary artery was enlarged 2 plus. The barium filled esophagus showed definite displacement posteriorly, indicating a 2 plus enlargement of the left atrium.

X ray findings confirmed the findings of fluoroscopy.

Laboratory data contained no contraindication to surgery. Hemoglobin was 15 Gm, 97 per cent, red blood count, 4.97 million per cubic millimeter, and white blood count, 5,500 per cubic millimeter. Polymorphs were 65 per cent, lymphocytes, 32 per cent, monocytes, 2 per cent, eosinophils, 1 per cent, bleeding time, 1 min, 18 sec, coagulation time, 4 min, 52 sec., prothrombin time, 20 per cent, 75 per cent of normal, hematocrit, 42 per cent, blood sugar, 129 mg per cent, blood urea nitrogen, 15.5 mg per cent, total proteins, 6 Gm per cent, albumin 2.15 Gm per cent, and globulin, 3.85 Gm per cent. Blood serology was negative by Wassermann and Kahn tests. Blood type was O, Rh positive. Urinalysis revealed a yellow, clear, acid specimen with specific gravity of 1.005, a faint trace of albumin, and no sugar. Microscopic examination of the urine revealed many epithelial cells and 6 to 8 white blood cells per high power field.

Preanesthetic consultation revealed a blood pressure of 115/80 in the right arm. Pulsations were felt over the major vessels except the left dorsalis pedis. It was felt that when prepared, this patient would be a fair risk for surgery, and accordingly the following premedication was suggested: Nembutal, 1½ grain the night before operation, Nembutal, ¾ grain two hours preoperatively, and atropine, grain ¼₆₀ when called to the operating room. The patient was to be sent to the operating room with three to four pillows under her head.

Preoperative Course—The history, clinical, electrocardiograph, and x ray findings pointed to a diagnosis of rheumatic heart disease, mitral stenosis, pericardial effusion, auricular fibrillation, with functional capacity class 3 C. The cardiologist felt that there was a question as to whether some rheumatic activity was present at this time since a pericardial effusion was present. For this reason, a period of enforced bed rest was advised. She was to be observed and digitalized before recommendation of surgery at this time.

On January 13, she had developed gradual numbness and weakness in the left arm and leg following an emotional disturbance. She also had unexplainable vomiting which was refractory to all measures three days prior to the onset of this hemiplegia. The episode of hemiplegia was treated by stellate block performed on the right side with Metrcaine, 1.5 per cent. She was also treated with anticoagulants, heparin and Danilone. By January 21, she was able to extend her left foot and toes well. However, dorsiflexion and rotation of the hip were poor. The left arm and hand were still weak and movements clumsy.

A conference was held with the Cardiologist on January 21. It was decided that because of the repeated cerebral emboli, surgery should not be delayed further. As the patient had been receiving heparin, this was discontinued on the following day.

On January 23 mitral commissurotomy was performed. Pertinent details from the operative report follow. With the patient in the right lateral position, the left hemithorax was opened through the fourth intercostal space. The pericardium was markedly distended with straw-colored fluid estimated at 500 cc. The pericardium was incised posterior to the phrenic nerve. A diastolic thrill was felt over the left ventricle. No systolic thrill was felt over the left atrium or pulmonary veins. The mediastinal vessels (left common carotid and unioinnate arteries) were isolated, and umbilical tapes passed around them. The free ends of the tape on the innominate artery were incorporated in a Rumel tourniquet. These vessels were knicked during such strategic moments as when the operator manipulated the left auricular appendage or during subsequent intracardiac maneuvers.

A purse-string suture of No. 2 braided silk was passed around the base of the auricular appendage. The free ends of the suture were fixed to a Rumel tourniquet. A Satinsky clamp was used to grasp the auricular appendage, and a 2 cm. incision was made at its tip. The appendage was thrombosed. The operator's little finger was introduced into the left atrium and the mitral valve could barely admit the tip. Owing to the failure of digital commissurotomy, both commissures were cut. As the operator's right little finger was withdrawn, a large clot approximately 3 to 4 cm. in diameter was palpated lying loosely within the left atrium. The clot was manipulated to the left atrial wall and was compressed between it and the little finger. A Kelly clamp was then used to bite the clot, and the finger, clot, and clamp were withdrawn in one motion. The blood loss before the appendage could be grasped between the thumb and forefinger was estimated at 300 to 400 cc., which was replaced.

The little finger was then withdrawn from the left atrium as the purse-string suture at the base of the auricular appendage was tied securely. A biopsy of the auricular appendage was submitted for histologic examination. The appendage was then oversewn with No. 00 cotton. The pericardium was left open for drainage. A chest tube connected to an underwater seal drainage bottle was brought out through the skin over the eighth interspace in the midaxillary line. Ribs four and five were approximated with four pericostal sutures. The chest wall was closed in layers and the skin was approximated with a continuous suture of No. 25 stainless steel wire. The second to the tenth thoracic vertebrae were infiltrated with 15 cc. of 2.5 per cent Neodolamine. The patient withstood the operative procedure well. Following recovery from anesthesia, she was able to move her limbs, although there was a transient sluggishness in the movements of her left leg. A stellate block on the right side was performed. The patient felt better, and was returned to her room in good condition.

Her postoperative course was essentially uneventful except for some pain in the calf of the right leg. In the twenty-four hours following surgery, this was treated conservatively and disappeared in the next twenty-four hours. Her chest tube was removed at the end of forty-eight hours, and the sutures were removed on the twelfth postoperative day. She was on a maintenance dose of digitoxin, 0.2 mg. once a day. Active movements of the feet were encouraged and physiotherapy was instituted during the entire hospital course.

She was finally discharged on Feb 11, 1954, and was advised to take digitoxin, 2 mg once a day

CASE 2—A 55 year old white housewife was admitted on March 30, 1954, with a questionable history of rheumatic fever as a child, and again at the age of 23 years, following her second pregnancy. At the age of 23 she was told that she had mitral stenosis, and at that time she noticed occasional precordial pains not requiring medication. Orthopnea had been present for about five years, and paroxysmal nocturnal dyspnea for two to three years. Onset of significant fatigue and dyspnea on exertion was two and one half years ago. Dizziness had occurred several times but no syncope. She had had hemoptysis on two occasions in 1939. She had no definite edema or ascites.

In September, 1953, an apparent episode of congestive failure had occurred, following which the patient was digitalized and given mercurials temporarily. Digitalis had been continued to the present time. She stated that she had been given quinidine only once preoperatively for her sympathectomy in 1939. She had frequent indigestion but denied a cough. She described an episode of arthritis in July, 1953, at which time she was given a three day period of treatment with cortisone. There was a history of possible embolization in September, 1953, with some symptoms in her left leg, but it was not certain whether this was central or peripheral. Significant associated history was Raynaud's disease treated with bilateral cervical sympathectomy in 1939, and bilateral lumbar sympathectomy in 1940 with apparent relief of her major symptoms.

Physical examination on admission revealed a blood pressure of 130/70 with atrial fibrillation. Examination of the heart revealed a sharp first sound and an apical midlate diastolic murmur. The pulmonic sound was accentuated 1 plus, the aortic sound was normal, and there were no other murmurs.

The abdomen had healed bilateral transverse lumbar scars and healed suprapubic scars with a small central diastasis. The liver was palpable one finger below the costal margin. There were no other masses or tenderness.

Otherwise, the physical examination was entirely normal.

Laboratory Reports—On admission, the prothrombin time was 90 per cent. Complete blood count, including bleeding and clotting time, was essentially within normal limits. Complete urinalysis was essentially normal, with a faint trace of protein. Total protein was 8.8, albumen, 5.2, and globulin, 3.6. Repeat urinalysis showed no evidence of protein in one specimen, and 2 plus on a second specimen postoperatively.

Electrocardiogram on March 31, 1954, showed auricular fibrillation with well controlled ventricular rate and minimal digitalis effect.

Preliminary x ray showed a circular area of calcification in the gallbladder area.

X ray of the Chest, Fluoroscopy, and Preliminary Cardiac Studies—The heart was slightly enlarged, configuration and chamber enlargement were consistent with mitral stenosis. Postoperative x rays showed moderate pleural reaction and good expansion of both lung fields.

Brachial artery tracing on April 7, 1954, showed no evidence of aortic stenosis or insufficiency.

Hospital Course—Weight on admission was 116 pounds with only a 6 pound loss on mercurial diuretics. She was afebrile on admission. After preliminary studies were completed, the patient was brought to surgery on April 9, 1954.

Pertinent Portions of the Operative Note—The pericardium was opened approximately 1 cm inferior and parallel to the left phrenic nerve. A purse string suture was placed in the base of the appendage and attached to a Rumel tourniquet. A Satinsky clamp was then applied to the base of the appendage, and the distal segment of the appendage was opened. The left atrial appendage contained both old and new clots peripheral to the area enclosed by the Satinsky clamp. After carefully irrigating and flushing the distal extremity of the left atrial appendage, the Satinsky clamp was removed for a few seconds, and it was permitted to bleed. As no clots washed out the auricle was apparently clear. Accordingly, a finger was inserted

but it was soon detected that a free clot approximately the size of a walnut was lying in the posterior extremity of the left atrial wall. The clot was compressed between the index finger and the lateral wall of the left atrium and manipulated to the appendage. A Kelly clamp was then placed along the index finger, the Bland tourniquet was loosened, and the clot was evacuated. Bleeding was controlled by digital compression of the appendage after the clot was evacuated. After this, commissurotomy was carried out by the instrumental method, opening the valve from a size less than a finger tip to one and one half fingers with the production of an estimated 4 c.c. of regurgitation. No aortic disease was detected by palpation.

Postoperatively, the patient developed a rapid atrial fibrillation rate up to 144 and was given additional doses of Cedilamid. She responded well, with the rate remaining around 100. Blood pressure was well maintained throughout. Both lung fields expanded well. There was no significant trouble with secretions. Intake and output were adequate. Chest drain tubes were removed on the second postoperative day.

Beginning on April 19, 1954, the patient developed a right upper quadrant distress and fullness in her abdomen with some tenderness in the liver area. There may have been some enlargement of the liver, but exact examination was difficult because of tenderness. All sutures were removed on April 20, 1954. Later this day she complained again of more severe abdominal pain. There was some elevation of temperature and some rebound pain and referred pain in the right upper quadrant. Urinalyses were essentially normal. Flat plates of the abdomen taken on April 22, 1954, suggested calculus in the gallbladder area, and cholangiograms were ordered.

Pathologic Report on Surgical Specimen (April 9, 1954).—Blood clot of recent origin, auricular appendage, myocardial fibrosis.

Fluoroscopy postoperatively showed no evidence of fluid collection. There was good mobility of both domes of the diaphragm. The right upper quadrant tenderness subsided, and temperature returned to normal range.

The patient was discharged improved on April 24, 1954.

CASE 3—A 49 year old white woman, who had had a questionable history of rheumatic fever at the ages of 7, 9, and 19 years, was admitted April 4, 1954. A heart murmur was known apparently only the past few years. She had a history of pneumonia at the age of 34 years, and had had dyspnea on exertion for the past ten years, more noticeably progressive during the past two years. Exercise tolerance was reduced to a limitation of a walk of $1\frac{1}{2}$ blocks or one flight of stairs. She had been hospitalized two years before for congestive failure and had been taking digitalis since. Mercurials averaged once per week for the past three years. She had had no quinidine. She had been on salt restriction for two years. Ankle edema has been intermittent for two years. She had had a sensation of fullness in her epigastrium, but no definite ascites or hemoptysis. She had had a nonproductive nervous cough for some time.

Physical Examination.—Blood pressure was 130/90. Examination of the heart revealed a soft, long diastolic murmur with a snapping first sound at the apex. A soft diastolic murmur, best heard in the left second, third, and fourth interspaces, was extremely faint in the aortic area. No systolic murmurs were audible. There were a few fine râles at the bases posteriorly with no wheezes or rhonchi, and no dullness. The liver was palpable one finger breadth below the costal margin. There was no edema of the lower extremities. There was no thrill palpable. The aorta was normal in size, but, at the base, there was a faint rough systolic thrill together with a distinct early diastolic thrill. The heart was enlarged predominantly in the right ventricle, which was enlarged about 2 plus with the left ventricle being slightly enlarged. The rhythm of the heart was regular although the myocardium was somewhat irritable on manipulation. The anterior descending artery was noted to be tortuous but not hardened. The left atrium was enlarged to about 2 plus, with the appendage enlarged to about the same degree. The appendage was fibrillating and was somewhat distended.

Over the left ventricle was felt a distinct diastolic thrill, but over the left atrium no thrill was noted. Any changes in this thrill and those palpated over the base of the aorta were not noted because of hypotension.

Laboratory Findings—Complete blood count, including bleeding and clotting times, and serology were normal on admission.

Bilirubin was 0.1 and 1 minute, total 0.4. Chlorides and sodium were normal preoperatively. In postoperative blood counts, red blood cells and hemoglobin were within normal limits.

Urinalysis showed proteinuria, but repeat blood counts were within normal limits.

X rays—Preliminary cardiac studies indicated left atrial and right ventricular enlargement consistent with acquired mitral valvular disease.

Electrocardiogram on admission revealed atrial fibrillation with well controlled ventricular rate. Digitalis effect was present. There was suggestion of right ventricular hypertrophy and strain.

Brachial artery tracing was performed on April 22 and showed an anacrotic notch, and some delay in systole suggested some degree of aortic stenosis.

Surgery was performed on April 26, 1954. Pertinent points follow. Findings showed the pulmonary artery was enlarged about 3 plus.

Operative Procedure—The pericardium was incised anterior to the phrenic nerve to give a better exposure of the left ventricle for the aortic dilatation. The left auricular appendage was then purse stringed with No. 3 Anacap silk on an Atraumatic needle and was then opened in the routine manner. At this point, the organized clot already mentioned was noted and after freeing some parts of it, the left atrium was flushed adequately to get rid of whatever thrombi could be flushed out. At this point, the finger was inserted and the mitral valve was explored. As previously mentioned, the mitral valve opening was less than a finger tip in size. After digital pressure, the anterior commissure was split, giving a resultant opening of $1\frac{1}{4}$ fingers. As previously mentioned also, a 2 cc regurgitation was felt posteriorly before the mitral commissurotomy. There was a resultant 4 cc regurgitation in the same area after the procedure. Following the commissurotomy, the examining finger felt a large fresh thrombus in the left atrium, which was carefully removed by clamping with a long curved forcep and pulling it while the purse string suture was fully loosened in order to flush it out together with some free thrombotic particles.

The patient's blood pressure then dropped to 80 systolic by palpatory methods with the pulse hardly perceptible. In the meantime, the purse string suture was tied, and the appendage was held from being oversewn until after aortic commissurotomy. As soon as the blood pressure rose to 110 systolic with corresponding improvement of the pulse the left ventricle was purse stringed about its apex where a dimple was felt. After making a stab incision in the purse stringed area, the beaded wire was inserted way beyond the aortic valve as felt by the left thumb and index fingers at the base of the aorta. The incision was then widened by further slitting with the knife, and the war head of the aortic dilator was inserted beyond the aortic annulus. Then, the valve was dilated three times by opening the aortic dilator. The dilator was then pulled out and the myocardial incision was repaired with mattress stitches of No. 000 cotton.

Incidentally, before the insertion of the dilator, two cross stitches of No. 000 cotton were placed opposite each other beyond the line of the purse string. These cross stitches were utilized during the closure of the myocardial incision. Remarkably this helped to prevent bleeding as usually encountered during the closure of the myocardial incision. Following the repair of the myocardium, it was noted that the heart was beating very feebly and slowly. At the same time the pulse was noted to be 60 per minute while the blood pressure was about 80 systolic. The left auricular appendage was then oversewn and preparation to close the chest as rapidly as possible was made in order that the patient could be turned on her back for the improvement of her pulse and blood pressure. Hemostasis was noted to be adequate as the chest cavity was flushed with saline. A thoracotomy tube was inserted in the ninth left lateral interspace and the ribs were approximated.

The chest was quickly closed. As soon as the patient was turned on her back, some improvement in her blood pressure and pulse was noted, and she was returned to her room in fair condition.

Postoperative Course—After surgery, the temperature spiked to 101° F rectally but returned to a normal range by the seventh postoperative day. Her postoperative course was extremely uneventful with the exception of slight abdominal distention. Chest tubes were removed by April 28. A few basal râles persisted, but cleared fairly well on coughing. The patient had a good aortic second sound postoperatively with a very soft systolic murmur in the aortic area on the left. No mitral murmur was present. A very early diastolic murmur became audible in the pulmonic area. The aortic second sound remained good throughout. All sutures were removed by May 7, with the wound healing well. Because of a slight weight gain the patient was given additional mercurial therapy.

She was discharged improved, on May, 11, 1954, with instructions to continue digitalis.

CASE 4—A 38 year-old white woman was admitted on July 28, 1954, with a history of known heart disease since her first pregnancy seventeen years before with precordial pain, hemoptysis, shortness of breath, and fatigue. The heart murmur was detected nine years prior to this present admission. Since January, 1954, she had hemoptysis and ankle edema on several occasions. She had three-pillow orthopnea and, for the past seven years, dizziness on several occasions. She had been on digitalis since 1944. There was nothing pertinent in the family history, but the father died of cancer of the liver.

Physical examination revealed a well developed, well nourished woman who appeared in no acute distress at the time of admission. Pertinent findings in this physical were confined to the chest. The chest was of normal configuration. It was symmetrical with equal expansion in both sides. Examination of the lungs revealed normal breath sounds and no adventitious sounds. Examination of the heart revealed auricular fibrillation with a rate of 96 per minute. The pulse was 80 per minute and the pulse deficit was 16 beats per minute. The blood pressure was 120/88. The systemic review was negative. On auscultation, there was a sharp mitral first sound with a mid-diastolic murmur at the apex. There was a slight systolic murmur over the aortic area with a good aortic second sound.

Fluoroscopy revealed a slender thoracic cage with clear lung fields, a 2 plus heart of mitral contour, no calcification in the aortic or mitral areas. There was a 2 plus outflow tract of the right ventricle. The left atrium was enlarged 2 plus. The pulmonary artery segment was 2 plus enlarged. The peripheral pulmonary vessels were not engorged very much. X-ray studies confirmed findings on fluoroscopy.

Laboratory data contained no contraindications to surgery. Hemoglobin was 12.1 Gm., white blood count, 8,500, polymorphs, 60 per cent, lymphocytes, 29 per cent, eosinophils, 1, stab forms, 2, hematocrit, 43 per cent, bleeding time, 1 min., 15 sec., coagulation time, 5 min., 25 sec., blood serology, negative, blood type, B Rh positive, blood urea, 18 mg per cent, total proteins, 7.2 Gm. per cent, albumin, 4.8 Gm. per cent, globulin, 2.4 Gm. per cent, and urinalysis, yellow, acid with specific gravity at 1.035 and a trace of protein but no sugar. The patient was digitalized for surgery and was fibrillating prior to surgery.

At the medical conference on July 30, 1954, a mitral commissurotomy was approved. It was felt that after the mitral valve had been opened, the aortic valve should be palpated to see if significant aortic stenosis was present. If this was slight, as seen on the brachial artery tracing and accompanied by a faint aortic systolic thrill it might be best left alone.

The patient was originally scheduled for surgery on Aug. 2, 1954, but the operation was cancelled in view of her rapid weight loss, presence of some sugar in her urine, and an elevated sedimentation rate and blood urine nitrogen. The lungs were clear. She was again scheduled on Aug. 6, 1954, and a mitral commissurotomy was performed.

Pertinent Portions of the Operative Note—The pericardium was opened anterior to the phrenic nerve. The left auricular appendage was thrombosed. A purse-string suture was placed around the base of the left auricular appendage and the appendage was opened. An old healed thrombus was removed from the auricular appendage. In addition, the Satinsky

SUMMARY

1 A maneuver for extracting a pedicled ball thrombus from the auricle is presented. This maneuver is comprised of three parts: (1) compressing the clot between the ungloved index finger and auricle, (2) extraction of the clot by finger traction and grasping with a Kelly clamp, and (3) getting rid of any loose particles by allowing the unclamped auricle to bleed freely during the time between pronation and supination of the hand.

2 Use of the fifth finger in entering a small or thrombosed appendage is described.

3 Application of the hemostatic cardiac mattress sutures to the performance of aortic commissurotomy is described.

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ADDENDUM

Since this article was submitted for publication, four consecutive additional cases have been operated on successfully using this maneuver to extract the clots from the atrium, with no deaths or complications from emboli.

GAS VOLUME CORRECTION TABLE FOR CARDIOPULMONARY LABORATORIES

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IN THIS communication the authors hope to save investigators in cardio pulmonary laboratories much tedious labor in their daily calculations by laying before the reader a table of corrections of gas volumes from various spirometer temperatures to body temperatures of 36° C, saturated with water vapor and at ambient barometric pressures

By the simplest mathematics one can deduce the following equations from the elementary gas law to correct for temperature variations in a dry gas

$$V_t = (1+0.00367 T) V_o$$

therefore

$$V_o = \frac{V_t}{1+0.00367 T}$$

$$V_b = (1+0.00367 T_b) V_o$$

substituting for V_o

$$V_b = \frac{1+0.00367 T_b}{1+0.00367 T} V_t \quad (\Lambda)$$

Where

V_t = volume of a dry gas at spirometer temperature °C

T = spirometer temperature °C

V_o = volume of gas at 0° C

V_b = volume of gas at body temperature of 37° C, dry

T_b = body temperature 37° C

Since the gases in both the spirometer and lungs are considered to be saturated with water vapor and since the vapor pressure of water is different at spirometer and body temperatures equation (Λ), corrected for this water vapor pressure difference becomes

$$V = \frac{(1+0.00367 T_b)}{(1+0.00367 T)} \frac{(P - P_t)}{P - P_{tb}} V_t$$

Where

V = volume of saturated gas at ambient pressure and body temperature

P = ambient barometric pressure

P_t = water vapor pressure at spirometer temperature

P_{tb} = water vapor pressure at body temperature

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TABLE I

TEMP (°C)	610	620	630	640	650	660	670	680	690	700	710	720	730	740	750	760	770	780
15	1.189	1.177	1.167	1.159	1.149	1.140	1.130	1.122	1.114	1.106	1.100	1.094	1.088	1.083	1.078	1.073	1.068	1.063
16	1.111	1.127	1.143	1.160	1.176	1.190	1.205	1.221	1.236	1.253	1.268	1.284	1.298	1.315	1.328	1.341	1.354	1.367
17	1.277	1.298	1.320	1.343	1.366	1.388	1.410	1.432	1.454	1.476	1.498	1.520	1.542	1.564	1.586	1.608	1.630	1.651
18	1.222	1.212	1.201	1.191	1.180	1.170	1.160	1.150	1.140	1.130	1.120	1.110	1.100	1.090	1.080	1.070	1.060	1.050
19	1.167	1.156	1.147	1.139	1.131	1.123	1.115	1.107	1.100	1.093	1.086	1.079	1.072	1.065	1.058	1.051	1.044	1.037
20	1.108	1.099	1.091	1.084	1.077	1.070	1.063	1.056	1.050	1.043	1.036	1.030	1.023	1.016	1.010	1.003	0.996	0.989
21	1.056	1.042	1.031	1.027	1.019	1.011	1.004	0.997	0.990	0.984	0.978	0.971	0.965	0.958	0.951	0.945	0.938	0.931
22	1.002	0.984	0.970	0.960	0.952	0.944	0.936	0.928	0.921	0.914	0.907	0.900	0.893	0.886	0.879	0.872	0.865	0.858
23	0.942	0.925	0.910	0.898	0.887	0.876	0.865	0.854	0.843	0.832	0.821	0.810	0.800	0.789	0.778	0.767	0.756	0.745
24	0.871	0.860	0.850	0.842	0.834	0.826	0.818	0.810	0.802	0.794	0.786	0.778	0.770	0.762	0.754	0.746	0.738	0.730
25	0.812	0.800	0.791	0.783	0.775	0.767	0.759	0.751	0.743	0.735	0.727	0.719	0.711	0.703	0.695	0.687	0.679	0.671
26	0.751	0.740	0.731	0.723	0.715	0.707	0.699	0.691	0.683	0.675	0.667	0.659	0.651	0.643	0.635	0.627	0.619	0.611
27	0.688	0.682	0.677	0.671	0.666	0.660	0.654	0.648	0.642	0.636	0.630	0.624	0.618	0.612	0.606	0.600	0.594	0.588
28	0.625	0.619	0.614	0.609	0.604	0.599	0.593	0.588	0.582	0.576	0.570	0.564	0.558	0.552	0.546	0.540	0.534	0.528
29	0.560	0.555	0.550	0.546	0.541	0.537	0.533	0.528	0.523	0.518	0.513	0.508	0.503	0.498	0.493	0.488	0.483	0.478
30	0.491	0.486	0.481	0.476	0.471	0.466	0.461	0.456	0.451	0.446	0.441	0.436	0.431	0.426	0.421	0.416	0.411	0.406

Therefore the correction factor F by which the observed volume of wet gas at a temperature T must be multiplied in order to give the corrected volume at body temperature of 37° C, saturated, and at ambient pressure is given by the expression

$$F = \frac{(1+0.00367 T_b)}{1+0.00367 T} \cdot \frac{(P - P_t)}{P - P_{tb}}$$

Table I has been calculated to give this correction factor for barometric pressures ranging from 640 to 780 mm Hg and for temperatures ranging from 15° to 30° C

SUMMARY

A table of corrections is presented to convert respired air volumes to the volume at body temperature saturated and at ambient pressure

THE USE OF EVANS BLUE TO OUTLINE THE COURSE OF THE THORACIC DUCT

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BROOKLINE MASS

AN INVESTIGATION of the more recent literature on chylothorax^{1 2 3 4 5 6 7 8 9 10} reveals only two human cases¹¹ in which a dye was used to color the lymph and in only one instance was it employed to facilitate the closure of a leaking thoracic duct. The practical value of a quick, effective, and simple method of outlining the course of the thoracic duct is emphasized by the possibility of accidental division of the duct during esophagectomy, thoracic sympathectomy, lobectomy, surgery of the great vessels and radical dissections of the lower neck. The probability of serious consequences following posttraumatic chylothorax has been well described by other authors.^{1 12}

Reports in the recent surgical literature on the management of posttraumatic chylothorax show that when conservative treatment by aspiration, drainage, and suction is unsuccessful surgical intervention is far less hazardous than formerly.

Cases in which the thoracic duct was successfully ligated have been described, and the criteria for surgical intervention have been presented.^{13 14}

Usually after accidental division of the thoracic duct, the watery fluid coming from the proximal end is easily observed. There are exceptional cases, however, in which the source of the leak is extremely difficult to find. It is desirable, therefore, to determine what dye could facilitate the surgeon's finding a leak in a ruptured thoracic duct.

While investigating the possibility of using dyes in the laboratory to outline the course of lymphatic vessels we learned that fat-soluble and water-soluble dyes had been used.^{15 16} Crandall¹⁷ fed 250 mg of Sudan IV (D6) in 50 c.c. of olive oil to a patient with a thoracic duct fistula, noted the appearance of a faint red tinge in the drainage after a latency of an hour and a half and observed the deepening and fading of the color over a period of approximately six hours. Lee¹⁸ noted a staining of the thoracic duct after an equal latency when he fed the same dye in cream preoperatively to cats. We discarded this method because of its latency and the poor contrast offered between the lymphatics and the structures surrounding them.

We studied Evans Blue (T 1824) (D56) a water soluble nontoxic dye used by Drinker and Yoffey¹⁹ to outline lymphatic vessels. A 1 per cent aqueous solution was tested in the laboratory via the intravenous, subcutaneous, and intraperitoneal routes (see Table I).

The 1 per cent aqueous solution of dye was made with distilled pyrogen free water and put up in sterile glass ampules containing 2 c.c. each.

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TABLE I LABORATORY FINDINGS, USING EVANS BLUE DYE TO OUTLINE THE THORACIC DUCT AND PARA AORTIC LYMPHATICS

ROUTE OF ADMINISTRATION	NO. OF ANIMALS	TIME UNTIL DYE APPEARED IN THORACIC DUCT	DURATION OF STAINING OF THORACIC DUCT	OPTIMAL DOSE	COLOR OF DUCT		
					LIGHT	DARK	NONE
Subcutaneous (in lower leg)	8	23 min	12 min	0.5 to 1.0 mg/kg		8	
Intravenous	17	17 hr	1 hr, 10 min where observed	3 mg/kg	5	7	5
Intraperitoneal	1	10 min	1 hr	3 mg/kg		1	

We found that by administering 0.5 to 1.0 mg per kilogram of body weight subcutaneously in the lower leg of dogs, cats, and monkeys, we successfully outlined the thoracic duct in all cases, and decided to use this method in two human cases.

DISCUSSION

It is well known that the walls of the lymphatic capillaries are permeable to certain dyes⁸ and Evans Blue is thus absorbed into the lymphatic system as a foreign substance, carried to the thoracic duct, linked to the albumin fraction of the serum proteins, and excreted via the gall bladder.

The use of this dye in 1 per cent aqueous solution injected subcutaneously into a lower limb to outline the thoracic duct has been universally successful in our hands if either the duct or one of its major trunks was not ligated. It was found that an effective dose was 1 mg per kilogram of body weight, beyond this dosage there was danger of producing widespread staining of the animal. In the human, doses of 0.7 to 0.8 mg per kilogram proved adequate. We feel however that a total dose of 25 mg should not be exceeded because of the possibility of deep, widespread staining of the patient's skin.

The advantages of this method are (1) reliability of the procedure, (2) ease of administration, (3) good color contrast between the thoracic duct and surrounding structures, (4) short latency between injection and appearance in the thoracic duct, and (5) the rapid fading of the stain from the injection site within approximately eight weeks after injection.

Evans Blue was used successfully in two cases. In one it helped to visualize the leak in the thoracic duct. In the other it determined whether or not the thoracic duct had been properly ligated after division. A third application of this technique would be the antiepileptory outlining of the thoracic duct to help the operator to avoid damaging it in dissection involving the esophagus, the thoracic sympathetic chain, the great vessels, the hilum of the lungs, and structures at the base of the neck.

SUMMARY

The practical application of a quick, simple, and effective method of outlining the thoracic duct in experimental animals and patients has been described. Two illustrative cases, one of recovery from traumatic chylothorax following successful ligation of the thoracic duct, have been cited.

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OPERATIVE CORRECTION OF COARCTATION OF THE AORTA IN INFANTS

REPORT OF TWO CASES

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IN THE recent literature there have been a number of reports of operative repair of coarctation of the aorta in infants¹⁻⁷ The optimal age for definitive operation is unquestionably the second decade of life⁸ It should be emphasized, however, that since some infants and young children will die of cardiac failure long before they reach the optimal age, concern for the fate of an early anastomosis should not preclude an operation of immediate and vital importance The following case reports confirm the observation that this condition is not an uncommon cause of cardiac failure in infants and demonstrate the improvement which may follow surgical repair of the defect

CASE REPORTS

Case 1—J A S, No 368093, a white male infant, was admitted to the University of Virginia Hospital Dec 29, 1953, because of rapid and difficult respirations His mother had had no complications during pregnancy or delivery His weight at birth was 3,618 grams When he was six weeks of age, the family first noted an inspiratory retraction of the lower costal margins and, in addition perspiration about his head, but not elsewhere

On admission to this hospital, the physical examination revealed a well developed and well nourished but very irritable 5 month old infant with a large asymmetrical head measuring 43 cm in circumference The area of cardiac dullness was increased, a moderate degree of inspiratory retraction of the lower end of the sternum was observed, and an occasional wheeze was present Roentgenograms of the chest revealed a marked increase in the cardiac size, the cardiothoracic ratio being 67 per cent (Fig 1, 1) An electrocardiogram revealed a cardiac rate of 150 per minute and a P R interval of 0.10 second, as well as T wave changes consistent with left ventricular strain The femoral pulses were absent and the blood pressure in both arms was 140/90 mm Hg The liver was palpable three fingerbreadths below the right costal margin Examination of the eye grounds revealed normal fundi The urine was slightly cloudy with a pH of 7.5 and examination for sugar, albumin and formed elements was negative The hemoglobin was 10.5 Gm per 100 cc and the hematocrit was 36 per cent The white blood cell count was 12,800 per cubic millimeter and the differential white count was normal

Three days after admission the child had a rise in temperature to 102° F rectally and was found to have a left otitis media which required chemotherapy and subsequent myringotomy Angiocardiograms done under sodium Pentothal anesthesia, revealed a normal aortic arch and brachiocephalic vessels although the arch ended rather abruptly below the origin of the left subclavian artery No lower aortic segment could be visualized (Fig 1, 2) The previous clinical diagnosis of coarctation of the aorta seemed to be confirmed

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Skin temperatures taken with a thermocouple are recorded in Table I.

On Jan. 19 1954 the patient was taken to the operating room. Following induction with cyclopropane anesthesia was maintained in a superficial plane with an endotracheal mixture of ether vapor and oxygen. The cardiac action was followed with the Cambridge Cardioscope*. During the operation the blood pressure ranged from 200 to 140 mm. Hg systolic and 100 to 90 mm Hg diastolic. After the chest had been opened through the fourth left intercostal space retraction of the lung anteriorly disclosed an obvious vascular abnormality in the gutter. On dissection of the mediastinal pleura a large left subclavian artery was identified arising far down the descending portion of the aortic arch.

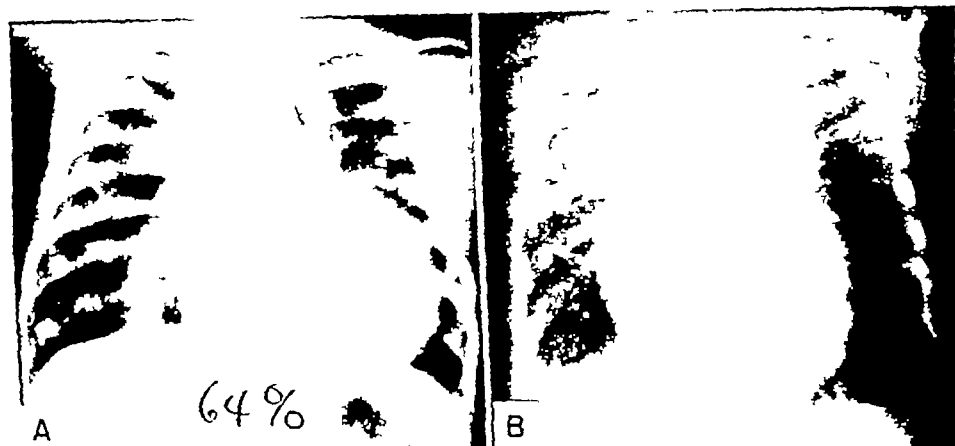


Fig. 1—J. A. S. A Posteroanterior roentgenogram. B Angiocardiogram in lateral anterior oblique position.

Several large intercostal vessels entered the lower aortic segment, which was separated from the upper segment by a marked constriction at the level of the fifth dorsal vertebra (Fig. 2). A palpable diaphragm could be detected at the site of greatest narrowing.

From the mid point of the coarctation medially, a ligamentum measuring approximately 2 mm. in diameter coursed forward to enter the posterior aspect of the left pulmonary artery. The lower segment though of normal size did not pulsate and was easily compressible. A small bronchial artery arising from the upper segment just proximal to the coarctation was divided between ligatures, as were two large intercostal arteries which converged to enter the lower aortic segment posteriorly in its upper 2 cm. of length.

TABLE I. THERMOCOUPLE SKIN TEMPERATURE READINGS (°F)

SITE	F H (CONTROL)	S V B			J A S		
		PREOP	POSTOP/REATIVE		PREOP	POSTOP 3 MO	
			2 DAYS	3 MO			
Forehead	95.0	100.0	96.8	96.0	97.0	95.2	
Chest	{ Back	—	100+	98.8	96.0	97.5	96.0
	{ Front	95.5	100+	98.0	95.8	97.7	96.2
Arm	{ Right	—	—	94.6	89.7	—	90.2
	{ L ft	92.0	—	95.0	90.1	95.0	93.0
Knee	{ Right	90.2	93.2	91.0	92.0	94.8	93.0
	{ L ft	90.1	93.9	90.2	92.0	95.0	92.8
Foot	{ Right	89.0	100.0	94.4	91.0	97.5	91.4
	{ L ft	89.0	99.2	95.5	91.0	95.7	92.4

*The Cambridge Instrument Co., Inc., Oxford, N. Y.

Mobilization was completed by division of the ligamentum arteriosum between ligatures. Potts ductus clamps were applied proximally and distally, and the area of coarctation was excised. Its lumen was barely sufficient to admit the tip of a silver probe (Fig. 2). The

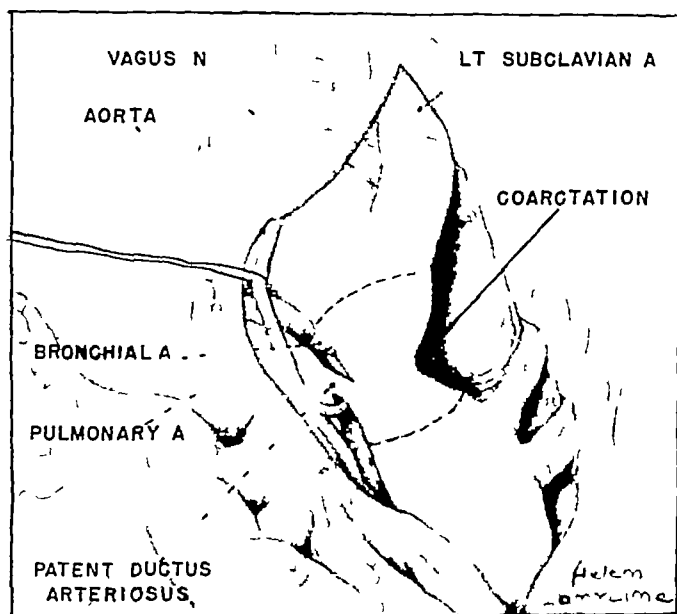


Fig. 2

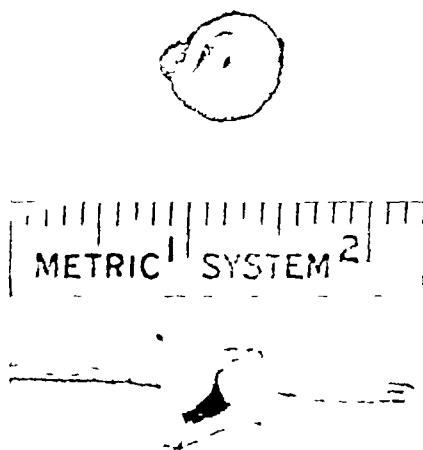


Fig. 3.—Narrowed segment of aorta resected from Case 1

ductus clamps were then approximated and a satisfactory end to end anastomosis was constructed with interrupted everting mattress sutures of 6/0 silk on Atraumatic needle. Upon cautious removal of the clamp, which had been in place a total of about forty-five minutes, the lower segment became taut and pulsated strongly.

TABLE II CLINICAL EVALUATION OF PERIPHERAL PULSES BY PALPATION

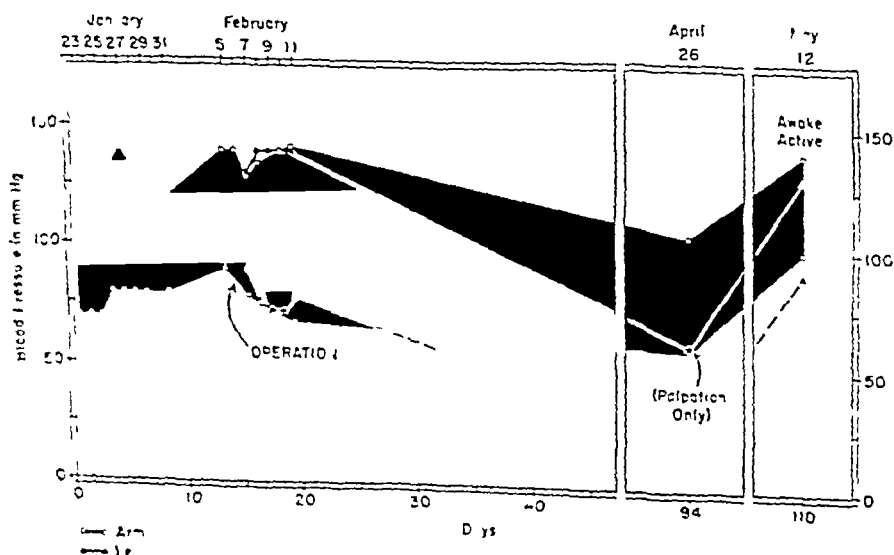
LOWER EXTREMITIES		S V B			J A S		
		PFEOP	POSTOPERATIVE		PREOP	POSTOPERATIVE	
			2 DAYS	3 MO		2 DAYS	3 MO
Femoral	{ Right	Trace (?)	++	4+	0	4+	4+
	{ Left	Trace (?)	4+	4+	0	4+	4+
Popliteal	{ Right	0	4+	3+	0	4+	3+
Posterior	{ Left	0	4+	4+	0	4+	3+
Tibial	{ Right	0	4+	3+	0	4+	3+
Dorsalis	{ Left	0	4+	4+	0	4+	3+
Pedis	{ Right	Trace (?)	4+	3+	0	4+	3+
	{ Left	Trace (?)	4+	4+	0	4+	3+

TABLE III CASE I (J A S) OSCILLOMETRIC READINGS IN UNITS OF OSCILLATION

3 MONTHS POSTOPERATIVE						
PRESSURE MM HG	ARMS		THIGHS		LEGS	
	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
170	00				00	
160	Trace	00	00		Trace	00
150	01	Trace	Trace	00	01	Trace
140	02	02	01	Trace	02	01
130	02	02	02	Trace	03	02
120	02	02	03	01	04	02
110	03	04	04	02	05	04
100	07	07	05	05	07	05
90	07	09	07	05	10	07
80	10	10	10	02	10	05
70	07	10	10	02	07	02
60	05	07	07	00	05	02
50	00	05	05		04	00
40		00	02		02	
30			00		00	

Chart I

J A S RECORD OF ARTERIAL BLOOD PRESSURES BEFORE AND AFTER OPERATION



The left pleural space was then washed with warm normal saline solution, and the lung was expanded with gentle positive pressure. The incision was closed, without pleural drainage, with interrupted fine silk sutures. At the conclusion of the operation the baby had received 150 cc of compatible blood. The blood pressure was noted to be 130/80 mm Hg and the breath sounds were equal over both lungs. A strong femoral pulse was palpated bilaterally. A postoperative roentgenogram of the chest revealed the left lung to be completely expanded.

Two days later the feet were warm, and pulses in the lower extremities were easily palpable (Table II). The sweating over the head and neck had ceased, the pronounced irritability present preoperatively was no longer evident, and the infant began to take his feedings well. Oscillometric readings are recorded in Table III. He was discharged on the tenth day after operation (see Chart I).

Case 2—S V B, No 366424, a 7 month old Negro girl, was admitted to the University of Virginia Hospital Jan 1, 1954 because of cough and fever of twenty four hours duration. She had been delivered in the same hospital on May 27, 1953 following an uncomplicated pregnancy. The birth weight was 3,459 grams, and no abnormalities were noted. She had had brief interval readmissions to the University Hospital at 5, 7, and 18 weeks for minor infections associated with dehydration and acidosis, and complicated by cardiac failure. In each instance she was treated by antibiotics, digitalization,



Fig 4—S V B 1 Posteroanterior projection B Angiocardiogram lateral anterior oblique position

and correction of fluid and electrolyte balance with appropriate therapy. Serial roentgenograms of the chest had shown progressive cardiac enlargement. On the present admission physical examination revealed an acutely ill infant who was quite small, weighing only 12 pounds, 12 ounces (5500 grams). Rectal temperature was 101.4° F, pulse was 175 per minute, and respirations were 88 per minute. Breath sounds over both lungs were thought to be decreased with a tubular quality high on the left anteriorly, and wheezes were present throughout. The liver and spleen were palpable one fingerbreadth below the costal margins. The blood pressure in both arms was 180/90 mm Hg. The femoral pulses were questionably palpable, and neither blood pressures nor pulses were obtainable in the lower limbs. A clinical diagnosis of coarctation of the aorta was made. Fluoroscopic and roentgenographic (Fig 4) examinations revealed a very large left ventricle. The laboratory work was essentially the same as on the previous admissions except for a normal

white blood cell count. The patient was treated with antibiotics, digitalization, and intravenous fluids and improved satisfactorily. Angiocardiograms showed a thick walled, somewhat dilated left ventricle, and a hypoplastic aortic arch. No dye was seen below the origin of the left subclavian artery (Fig 4 B). Skin temperatures (Table I), oscillometric readings, and blood pressure in arm and leg (Chart II) are summarized in Table IV.

Chart II

S.V.B. RECORD OF ARTERIAL BLOOD PRESSURES BEFORE AND AFTER OPERATION

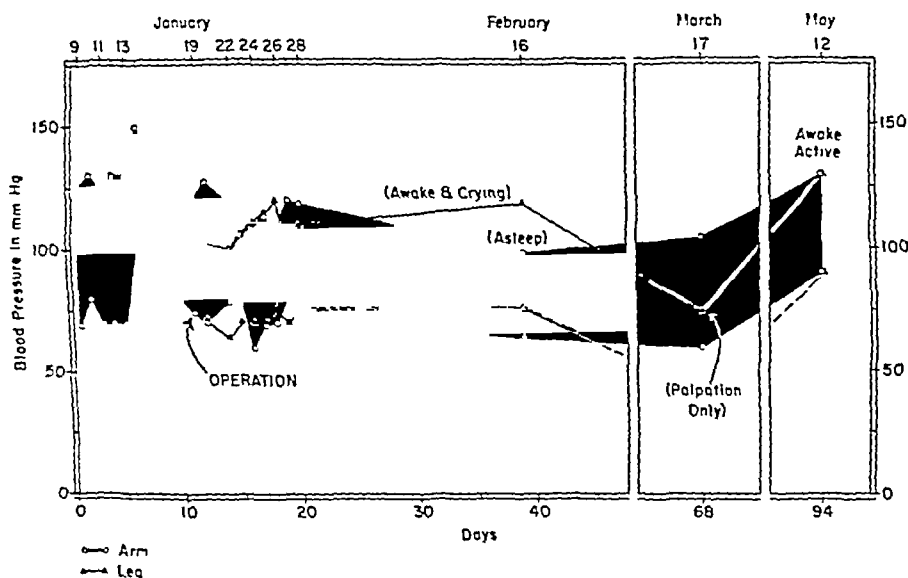


TABLE IV CASE 2(S.V.B.) OSCILLOMETRIC READINGS IN UNITS OF OSCILLATION*

IRES SUPE MM HG	RIGHT ARM			RIGHT THIGH			RIGHT LEG		
	PREOP	POSTOPERATIVE 2 DAYS	3 MO	PREOP	POSTOPERATIVE 2 DAYS	3 MO	PREOP	POSTOPERATIVE 2 DAYS	3 MO
230	0.0			0.0			0.0		
220	0.5			0.0			0.0		
210	0.5	0.0	0.0	0.0			0.0	0.0	0.0
200	0.5	0.2	Trace	0.0		0.0	0.0	Trace	Trace
190	0.5	0.2	Trace	0.0		0.0	0.0	Trace	Trace
180	0.5	0.4	0.2	0.0	0.2	Trace	0.0	0.2	0.1
170	0.5	0.5	0.4	0.0	0.2	0.3	0.0	0.7	0.4
160	0.4	0.4	0.5	0.0	0.2	0.4	0.0	1.0	0.5
150	0.3	0.4	0.5	0.0	0.5	0.5	0.0	1.0	0.5
140	0.3	0.4	0.8	0.0	0.2	0.7	0.0	0.7	1.0
130	0.3	0.2	1.0	0.0	0.2	0.4	0.0	0.7	1.0
120	0.2	0.1	1.0	0.0	0.2	0.2	0.0	0.5	0.7
110	0.2	0.0	1.0	0.0	0.0	Trace	0.0	0.2	0.7
100	0.2		0.9	0.0		Trace	0.0	0.2	0.7
90	0.0		0.7	0.0		Trace	0.0	0.2	0.5
80			0.4	0.0		0.0	0.0	Trace	0.2
70			0.3	0.0			0.0	0.0	Trace
60			0.2	0.0			0.0		0.0
50			0.0	0.0			0.0		0.0

* Values from the left extremities are proximate those recorded and are omitted for brevity.

On Feb 6, 1954, the patient was operated upon. Anesthesia was conducted in a manner similar to that of Case 1. Rectal temperature was measured by means of a thermocouple and maintained between 88° and 94° F by adjusting the flow of ice water through a Davol mattress*. A heavy collateral arterial circulation was present in the extracostal muscles. The pleura was entered through the fourth intercostal space. The lung was retracted forward and downward and dissection begun in the area where the coarctation was suspected. From the origin of the left common carotid artery distad there was a definite hypoplasia of the upper segment. Immediately below the origin of the left subclavian artery, a mild constriction of the aorta was succeeded by a bulbous expansion. This in turn was connected with a commodious lower segment by a narrow coarctation 3 to 4 mm in length (Fig 5).

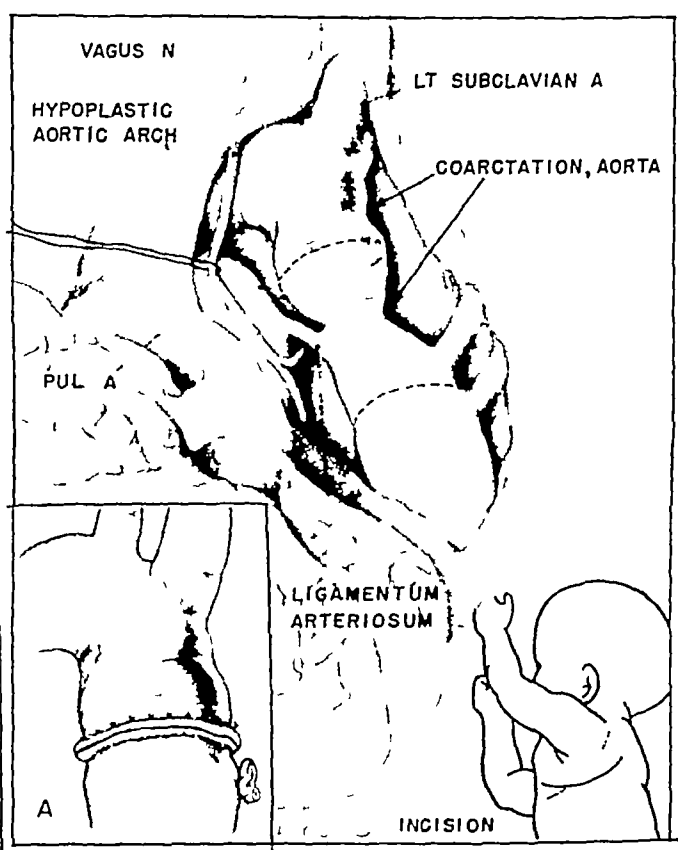


FIG 5

Strong pulsations were palpable throughout the upper aortic segment down to the point of true coarctation, below which very feeble pulsations and a strong thrill could be detected. The ligamentum arteriosum left the coarctated area and coursed sharply upward and forward into the posterior portion of the first part of the left pulmonary artery. This structure was doubly ligated with heavy silk; on division it appeared to contain a small but patent lumen. Three large intercostal arteries were divided between ligatures as they entered the lower segment, thus allowing satisfactory mobilization. The area of

*The Davol Rubber Co. Providence, R. I.

coarctation and the upper segment were carefully dissected from the surrounding areolar tissue. Two Potts ductus clamps were applied to the aorta above and below the coarctation, which was then excised (Fig 6). An anastomosis, measuring approximately 7 mm. in diameter, was accomplished with everting horizontal mattress sutures of 5-0 silk on Atraumatic needles. The procedure required approximately half an hour, and at the conclusion strong pulsations could be palpated in the lower segment and the thrill had completely disappeared. After lavage of the pleural space with normal saline solution the lung was expanded by gentle positive pressure and the incision was closed with interrupted sutures of fine silk.

At the beginning of the operation the blood pressure was noted to be 210 mm. Hg systolic and the pulse was 140 per minute. Throughout the procedure the systolic blood pressure ranged around 140 mm Hg, and the pulse was between 160 and 180 per minute. The moderate blood loss was replaced with 100 cc of blood. Weak femoral pulses, which

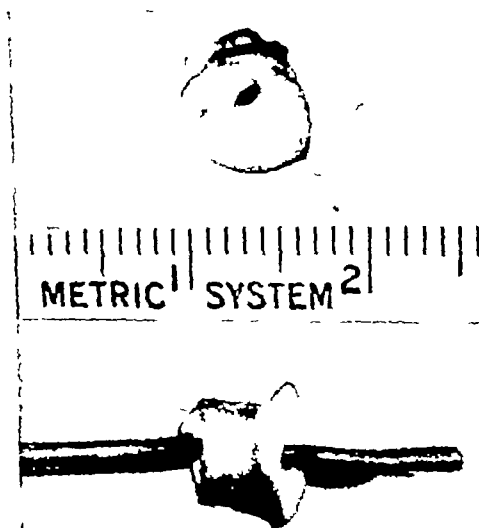


Fig 6—Narrowed segment of aorta resected from Case 2

prior to operation had been questionably obtainable, could now be palpated. Breath sounds were equal over both lungs. Shortly after the child returned to the ward, strong and full popliteal, posterior tibial, and dorsalis pedis pulses were palpated (Table I). On the first postoperative day the rectal temperature reached a peak of 101.6° F but thereafter was within normal range. Convalescence was uncomplicated, and she was discharged on the tenth postoperative day.

The child was seen as an outpatient April 26, 1954. The mother stated that she had a good appetite and had been well and playful. Fluoroscopic examination suggested that the cardiothoracic ratio was smaller than it had been preoperatively. Data from this and a subsequent visit are summarized in Table IV and Chart II.

Both patients were seen last on May 12, 1954. In each case the parents volunteered the information that the children had been much improved since their operations. Both were eating well, a fact which was substantiated by their gain in weight. S. V. B. weighed 7,020 grams as compared to a preoperative weight of 6,067 grams. J. A. S. weighed 9,000 grams as opposed to a preoperative weight of 5,973 grams. Neither patient was irritable or easily upset, and it was felt by both the parents and the physicians that the children appeared normal in all respects for their ages.

DISCUSSION

One of the common causes of cardiac failure in infants is coarctation of the aorta. Early failure is undoubtedly precipitated by common infectious processes which entail fever, elevation of pulse, and dehydration. Indeed the respiratory signs and symptoms of cardiac failure may easily be confused with a lower respiratory infection. In addition to these signs, however, the presence of hepatomegaly, an obvious increase in the size of the heart itself, the absence of femoral pulses, and an elevation of arterial pressure in the arms will confirm the diagnosis for the suspicious examiner.

Measurement of the blood pressure in the infant is not without difficulties, it requires considerable patience since pressure readings fluctuate widely with physical activity. The "flush" technique and the palpatory method give no information about the diastolic level. For the auscultatory method the smallest cuff (5.5 by 2.5 cm)* is satisfactory for the newborn only and we believe that the readings obtained with it are deceptively high. For infants up to 1 year of age a rubber cuff measuring 5 by 8 cm is more trustworthy. It should be applied snugly and taped in place. When the artery has been carefully located by palpation, auscultation is best done with a stethoscope which has a small bell. An ordinary bell-type stethoscope from which the Bakelite end piece has been removed may be lightly applied to the point of maximum pulsation.

Whether or not acute myocardial decompensation constitutes a surgical emergency is a question which can only be settled by longer experience. It seems obvious that progressive increase in size of the infant heart due to coarctation is ominous, particularly when cardiac failure has occurred. Under these circumstances digitalization and operative repair seem indicated.

Following operation in each of the above cases, the clinical relief was difficult to exaggerate. Prior to operation both infants were exceedingly irritable, thin, and distressed by common infections; moreover both had been in cardiac failure on two or more occasions. As early as the third postoperative day each child was relatively placid. Increase in appetite and gain in weight proceeded uninterrupted. Despite the disappointingly small reduction of blood pressure (Charts I and II) and the slight decrease in cardiothoracic ratio there has been no further evidence of cardiac failure. Should observation over the course of several years establish that the anastomotic site has failed to enlarge in proportion to the adjacent segments a second operation may eventually be required. But in this event these children will have been given an increased period of time in which to develop adequate collateral circulation. For the time being they are vastly improved.

CONCLUSION

Two detailed case reports have been presented of infants 18 and 30 weeks of age respectively in whom surgical repair of coarctation of the aorta has been successfully carried out. The indications were progressive cardiac en-

*W. A. Baum Company, Inc., Copeland, Long Island, N. Y.

largement and repeated myocardial decompensation. Attention has been called to the singular remission of certain symptoms despite the failure of marked fall in blood pressure.

We wish to acknowledge the assistance of Dr Harold F. Chase, Anesthesiologist in Charge, Department of Anesthesiology, University of Virginia Hospital, and of Dr Patricia Andrews, Anesthesiologist, University of Virginia Hospital, who administered the anesthesia.

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CANCER IN THE THORACIC STOMACH

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WARWICK, ENGLAND

THE development of carcinoma in a thoracic stomach is unusual and the recorded cases are, in most instances, cancers of the cardia in partial thoracic stomachs. Smithers,¹ reviewing the literature up to 1950, collected eleven, including six personal cases, and advanced three possible explanations for the association of the two diseases. Since this review of the subject, cases have been described by Dawson and Richards², Pack, Butler, and Katz³, and Robb.⁴ The incidence of the two diseases occurring together is unknown and their relationship to each other uncertain. Cancer of the body of the stomach developing in an organ entirely within the thorax is a rarity, and the case to be described has features of sufficient interest to justify its being recorded.

CASE REPORT

Mrs S S, aged 73 years, was admitted to the hospital under the care of Dr R D C Johnstone, in April, 1954, suffering from anemia of acute onset. Twenty nine years previously, she had undergone a radical mastectomy for carcinoma of the right breast. In 1944, she had a repair of an umbilical hernia. During the course of this operation it was found that she had a paraesophageal type of hiatus hernia. The stomach had passed up into the chest but could easily be drawn back into the abdomen. It was otherwise normal. In view of the patient's age, poor general condition, and the fact that the hernia had a wide neck, it was decided that repair of the diaphragmatic hernia should not be attempted. The umbilical herniorrhaphy was completed, and the patient made a good recovery. She remained well with no symptoms from the diaphragmatic hernia until ten days before her admission to the hospital. She then complained of giddiness and lassitude. The patient was well nourished, active, and well preserved. Apart from obvious anemia there were no physical signs. The blood pressure was 170/100.

Investigations

Blood count Hemoglobin 5.9 Gm per 100 c.c. (40 per cent), red blood cells, 4,000,000 per cubic millimeter.

Blood uric acid 45 mg per 100 c.c.

Fcccs Spectroscopic test for blood positive.

Fractional test meal Blood present in all specimens. No free hydrochloric acid.

Radiological examination A radiograph of the chest showed no abnormality. The report on a barium swallow read as follows: "A large irregular shallow crater anterior wall middle third of the thoracic stomach extending into the prepyloric antrum. Possibly a chronic peptic ulcer but highly suggestive of an ulcerating growth." (Fig 1)

It seemed certain that the source of bleeding was the ulcerated area in the stomach. The patient was given 5 pints of blood during the first week of her stay in hospital without detectable effect on her hemoglobin level. There was no vomiting. Operation was advised, and a further 5 pints of blood were given in the few days prior to operation. She was operated on sixteen days after admission to hospital, with a hemoglobin of 12.6 Gm. per 100 c.c. (85 per cent).

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Operation—With the patient in the lateral position, the chest was opened through the bed of the resected eighth left rib. The sac was readily seen extending upward from the esophageal hiatus. No adhesions between sac and lung were present. Within the sac a hard, craggy mass could be felt. The sac was opened, and the stomach was found within, it was free of adhesions and contained the large, hard ulcer. No other viscus was present in the sac. The length of the mesenteries of the stomach were such that it could be delivered further into the wound or returned into the abdominal cavity with ease, and the first part of the duodenum was noted to be actually touching the last part of the esophagus.



Fig 1—The carcinomatous ulcer is seen as a barium filled pear shaped opacity overlying the stomach shadow at its pyloric end (which is to the patient's left side)

A few small glands were present in the mesentery along-side the growth. It seemed neither necessary nor desirable to open the abdominal cavity to continue the exploration or to effect the anastomosis after total gastrectomy. Total gastrectomy was performed and the esophagus anastomosed to the first part of the duodenum from above the diaphragm, without division of the diaphragm or mobilization of the duodenum. The esophagus was of normal length. A two-layer end to end anastomosis using silk sutures was carried out, and a sleeve of peritoneum fashioned from sac was used to surround this junction. The lung was re-inflated and the chest closed without drainage. The stomach (Fig 2) was examined immediately following operation. The ulcer was clearly the source of the bleeding. Its base was full of friable blood clot. Removal of this produced a number of areas from which blood oozed. No single vessel could be found and no other macroscopic abnormality was present in the whole of the stomach.

Postoperative Progress—The patient's condition gave no cause for anxiety throughout. She received no pants of blood during the day of operation. She was up in the ward on

the third day. The test for occult blood in the feces became negative for the first time on the tenth postoperative day, and the patient was discharged to convalescence on the fifteenth day. A barium swallow showed no holdup at the anastomosis. Before and during the operation, the patient received a total of 13 pints of blood.

Pathology—The specimen consisted of an excised stomach some 18 cm in overall length. At the distal end there was a large crateriform ulcer 5 cm in diameter. It was firm, had a sloughing base and overhanging edges. There was a carcinoma of the gastric mucosa which infiltrated deeply into the muscularis. There was considerable necrosis toward the lumen. Six lymph nodes were examined without clear evidence of invasion.



Fig. 2—Stomach showing the carcinoma. The base of the ulcer was filled with friable blood clot. No blood vessel is visible in the base.

DISCUSSION

Various views on the possible relationship of the diaphragmatic hernia and the carcinoma have been put forward to explain the double pathology. Smithers¹ stated, "We must await the publication of accounts of further cases before we may draw conclusions as to the possible reasons for the development of cancer in these cases." Paek and associates³ admitted there was insufficient evidence to consider ectopia of the stomach a precancerous state. Belsey expressed the opinion that the relationship of carcinoma of the stomach and hiatus hernia occurs no more frequently than can be explained by the coincidence of two common diseases. The present case would appear to be in the category of a fortuitous association of the two diseases. The stomach had been herniated into the thorax for at least ten years and seemed an intrinsically normal stomach at the time of the patient's operation in 1944. There was no esophageal shortening and the possible effect of gastric juices in the development of cancer at the gastric cardia in patients with sliding hernias of the stomach is not relevant to growths of the body of the stomach. It is possible that such factors as reflux of gastric

juice, the production of esophagitis and ulceration of the lower end of the esophagus may be significant in the development of carcinoma at the esophago-gastric junction in patients with sliding hernias of the stomach. These factors do not influence the mucosa of the body of the stomach.

As well as this possible difference in etiology there may be a difference in the ease with which the condition can be treated surgically. The total gastrectomy performed in the case described was not as difficult as seemed probable from a preoperative study of the circumstances. In spite of the fact that the hernia was of long standing the abdomen had been previously opened and the stomach contained a large ulcer there was a complete absence of adhesions between the stomach and the sac and the sac and the intrathoracic viscera. The stomach was mobile the mesenteries long and the first part of the duodenum lay directly adjacent to the lower end of the esophagus. No mobilization of either esophagus or duodenum was necessary for the establishment of a tension-free esophagoduodenostomy. In their paper Pack and associates³ describe an almost identical case. The stomach lay in the chest and a large ulcerated carcinoma was present. The total gastrectomy was performed because of severe bleeding and they emphasize the ease with which the operation was carried out. They state that "the total gastrectomy performed within the chest was done with the greatest ease and speed in our experience. Their case was described as the first recorded instance of a total gastrectomy and esophagoduodenostomy done entirely from within the chest. It can be said of the para-esophageal hernia that the stomach as a rule is free in the sac and the fixity of the sac itself is exceptional. In contrast to this an operation for cancer of the cardia developing in a sliding hernia is a severe procedure. Delay in diagnosis, preoperative blood loss, the early involvement of vital structures, adhesions and perihatal fibrosis contribute to the severity of the operation. Accordingly the results of surgery in the recorded cases of cancer of the cardia in sliding hernias are for the most part disappointing. The presumption that operation in every case of cancer in a thoracic stomach will present technical difficulty and seriously disturb the patient is not justified, however.

I should like to thank Dr. M. Ierselski for the x-ray report.

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PULMONARY INFARCTION FOLLOWING INTERRUPTION OF THE PULMONARY ARTERY

REPORT OF A CASE

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IT HAS long been known that ligation of the pulmonary artery to a portion of lung will not of itself cause infarction. A review of the literature and excellent studies on the subject were reported in 1924 by Schlaepfer.¹ It was shown that the bronchial artery alone is normally sufficient to maintain the viability of the lung in animals. These findings were later corroborated by others such as Liebow and associates.² That these principles hold true in man can be assumed from the fact that the pulmonary artery may be ligated in the first stage of a two-stage pneumonectomy without complication.³ Indeed it is because it is felt that infarction is an unusual occurrence following ligation of the pulmonary artery that the following case is presented.

CASE REPORT

P. A. (8780), a 69 year old white man, was admitted to the Veterans Administration Hospital, Providence, R. I., on Dec. 6, 1951, with the chief complaint a weight loss of 40 pounds in six months. The patient had smoked two packages of cigarettes a day for many years. He claimed that he had always had a cough but that it had become more intense and productive during the past two to three years. No other cardiorespiratory symptoms were noted. Three months prior to admission, following chest x-ray the patient was told that he had a 'growth' on the lung. Past history was noncontributory.

Examination on Admission—The temperature was 99.4° F. pulse, 130 respirations 20 blood pressure, 160/90 mm. Hg. The patient was a fairly well developed, undernourished man who appeared chronically ill. There was a moderate increase in the anteroposterior diameter of the chest. Dullness to percussion and bronchovesicular breathing were present over the apex of the right lung. The remaining lung fields were hyperresonant to percussion. The heart sounds were distant and P was accentuated. There were diminished pulsations in the arteries of the right lower extremity. There was no evidence of peripheral edema.

Laboratory Findings—X-ray of the chest (Fig. 1, 4) revealed a hazy, homogeneous density in the right upper lung field with shift of the trachea to the right. The heart did not appear enlarged. Blood count on admission showed a white blood count of 24,600, 69 per cent segmented neutrophils, 13 per cent stab cells, 15 per cent lymphocytes, 3 per cent monocytes, hemoglobin 13.0 Gm. urinalysis was negative except for albumin 1 plus and 2 to 4 white blood cells per high power field. Sputum was negative for acid fast bacilli on smear. Preoperative and postoperative electrocardiograms revealed changes interpreted as evidence of old myocardial infarction.

Hospital Course—Bronchoscopy was negative. Sputum cytology showed no evidence of malignancy. Ventilatory studies showed maximum breathing capacity, 4871 liters per

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minute walking minute volume 14.94 liters per minute. Because the most likely diagnosis was carcinoma of the right upper lobe, it was decided that exploratory thoracotomy should be undertaken. It was felt that, if possible, a lobectomy would be performed.

On Dec. 20, 1951, the patient was taken to the operating room. The right upper lobe was consolidated and firmly adherent to the chest wall. By blunt and sharp dissection it was freed extrapleurally. As right upper lobectomy was being completed, a tumor was found close to the level where the right upper lobe bronchus was transected. Frozen section revealed epidermoid carcinoma. Pneumonectomy was therefore decided upon. Accordingly the main pulmonary artery was dissected free and divided. At this stage of the procedure the patient's condition deteriorated, and because of this, the operation was terminated.

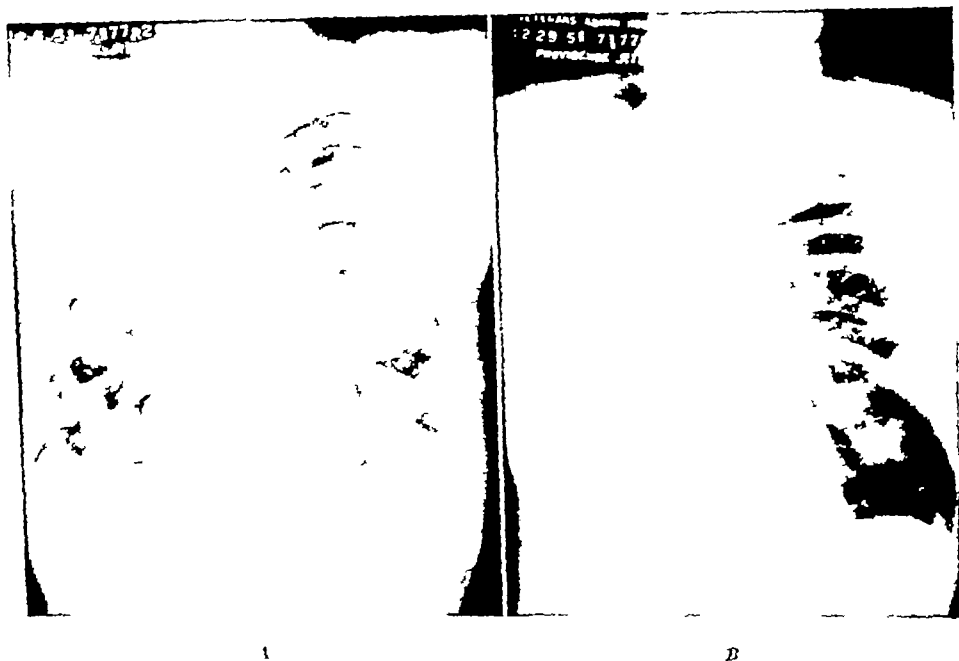


Fig. 1—A, Chest x ray on admission showing atelectatic right upper lobe. B, x ray taken on the sixth postoperative day. Opacity is almost entirely due to intrapulmonary clots.

Postoperatively, aqueous penicillin and streptomycin were given. The patient was encouraged to cough frequently, and expectorants were administered. Endotracheal suction was freely employed.

Following operation the patient experienced a febrile course with a maximum rectal temperature of 102.2° F. On the second postoperative day the patient started to cough up dark bloody sputum which gradually increased in volume. Concomitantly, the right lung field became increasingly opaque by x ray (Fig. 1, B). Rhonchi, especially over the right chest, became more numerous. The patient's condition gradually deteriorated until his death on the tenth postoperative day.

Post mortem examination revealed the following pertinent findings. The right thoracotomy wound was well healed. The right pleural cavity was obliterated by fibrous adhesions. The right pulmonary artery had been completely divided. The right upper lobe bronchus had been transected and sutured with fine silk. There was no evidence of tumor. The branches of the superior vena to the right upper lobe had been ligated and divided. All the bronchovascular branches to the middle lobe and right lower lobe were intact. The

right middle and lower lobes were red, dense, and firm to palpation. The left lung was voluminous, crepitant, and the cut surfaces were irregularly honeycombed. The trachea and the bronchi to both lungs were distended with bloody fluid, and their surfaces were hyperemic. The heart weighed 450 grams. It was increased in size especially in the left ventricular portion. Sectioning revealed scarring of the posterior ventricular wall. The coronary arteries were markedly sclerotic. The spleen weighed 125 grams. The liver weighed 2,250 grams. The lymph nodes were free from tumor.

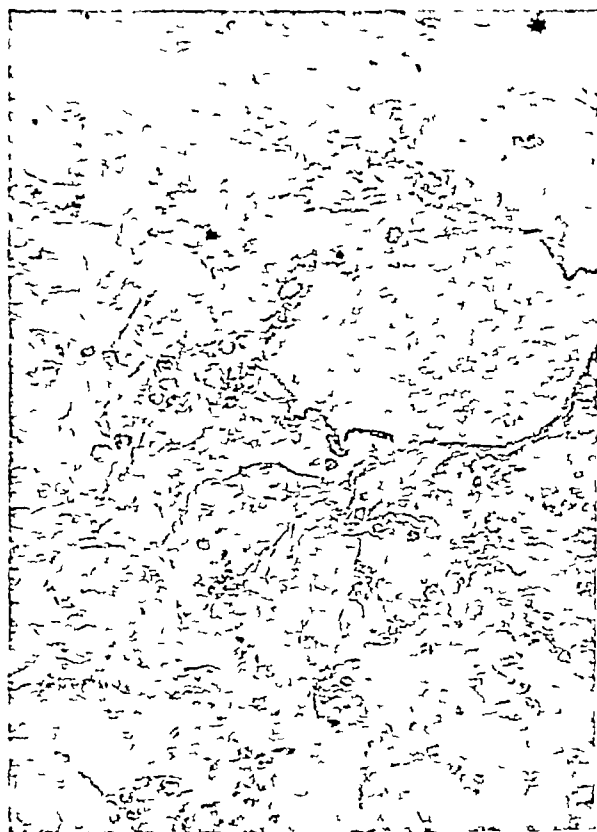


Fig 2—Photomicrograph of a portion of the right lower lobe showing the infarcted area

Microscopic sections of the right lung (Fig 2) revealed a massive infarction consisting of homogeneously staining, "ghostlike" structures. The alveoli were filled with disintegrating red cells, leukocytes, and hemosiderin-laden macrophages. Numerous bacteria were present. Sections of the liver revealed atrophy of the central two thirds of the lobules with distention of the veins and sinuses with red blood cells.

Post mortem culture of the lung produced predominantly *B. coli* with a few colonies of *A. aerogenes* and microaerophilic streptococcus. Sections of the tumor of the right upper lobe revealed anaplastic epidermoid carcinoma.

DISCUSSION

Several possible explanations might be offered for the occurrence of pulmonary infarction in the case presented. It might be postulated that the

bronchial circulation to the right middle and lower lobes had been compromised. However, this is not likely since the right main and intermediate bronchi had not been dissected free. Furthermore, it has been shown repeatedly^{1,2} that it is very difficult to compromise the bronchial circulation without completely interrupting the bronchus supplied since the bronchial vessels ramify not only over the external surface but also in the submucosa of the bronchi. Cudkowiez and Armstrong⁶ have recently shown that arteriosclerotic changes may occlude the bronchial arterial lumen in some instances of pulmonary emphysema. That this mechanism was not operable in this case may be inferred from the appearance of the arteries in the wall of the bronchus shown in Fig. 3.



Fig. 3—Photomicrograph of the bronchus to the right upper lobe. Two small arteries are shown.

Karsner and Ash⁷ long ago showed that venous congestion superimposed upon pulmonary arterial occlusion will produce infarction. That this mechanism might have at least been a contributory factor in this case is a distinct possibility, as post-mortem evidence such as the enlarged left ventricle and the central atrophy of the liver indicate. However, no clinical signs of congestive failure were evident prior to death.

One other possible contributory factor might be mentioned. Mathes and associates⁵ and Tuttle and Nicoll⁸ showed that, while ligation of the pulmonary artery alone will not produce infarction, early superimposition of infection may produce the necrosis.

SUMMARY

A case of pulmonary infarction following ligation of the pulmonary artery is presented. Possible mechanisms responsible for the production of the infarction are discussed.

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PRIMARY CYST (MESOTHELIAL) OF THE DIAPHRAGM

REPORT OF A CASE

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PRIMARY cysts of the diaphragm are rarely reported in the literature. In 1950, Samson and Childress¹ reviewed the literature on primary tumors of the diaphragm. Of 35 tumors recorded only three were cysts. In 1949 Aufses and Oseasohn² reported a mesothelial diaphragmatic cyst and stated that they were able to find only five other recorded cases. In 1951 Cruickshank and Cruickshank³ reviewed the literature and reported two diaphragmatic mesothelial cysts of their own bringing the total to eight primary cysts of the diaphragm. We wish to record another.

CASE HISTORY

Z. A., a 57 year old Negro man, was admitted to the hospital on Oct. 12, 1951, complaining of recurring attacks of left upper quadrant abdominal pain, pain the right shoulder and right infraclavicular regions and shortness of breath. All symptoms were precipitated by exertion and alleviated by bed rest. At times he complained of a dry, nonproductive cough. There was no hemoptysis. These symptoms had been present for twenty years.

Roentgenograms and fluoroscopy of the chest revealed a soft tissue tumor mass in the left leaf of the diaphragm which moved synchronously with the diaphragm. An old roentgenogram of the chest, taken elsewhere in 1931 for similar complaints, was available and showed the same mass present, the size having increased moderately in twenty years. At this time the patient refused further diagnostic procedures and left the hospital.

In the next two years he was admitted twice with similar complaints. He was readmitted in September 1953, complaining of severe upper left quadrant abdominal pain and dyspnea.

Physical examination revealed a tall, thin, asthenic Negro man, 57 years of age, who did not appear to be acutely ill. The blood pressure was 118/84. The heart was normal. The chest moved symmetrically, expansion was equal on both sides, and the lungs were

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Fig 1



Fig 2

Fig 1—Film taken in 1931 showing soft tissue tumor mass in left leaf of diaphragm

Fig 2—Film taken in August 1953 showing moderate increase in size of the tumor



Fig 3—Diagnostic pneumoperitoneum (September 1953) showing the tumor to be intra diaphragmatic

normal to percussion and auscultation. There was slight tenderness on deep palpation in the upper left quadrant of the abdomen with increase in pain on compression of the lower rib cage. No other abnormalities were noted.



Fig 4

Fig 5

Fig 4—Appearance of diaphragmatic cyst at surgery before excision.

Fig 5—Film taken at surgery showing defect in diaphragm after excision of cyst. Inferiorly the diaphragmatic peritoneum is intact.



Fig 6—Appearance of cyst after excision.

On bed rest all symptoms quickly subsided. There were no abnormal laboratory findings. Electrocardiogram, gastrointestinal series, and barium enema were normal. A broncho-copic examination was negative for pathology. A diagnostic pneumoperitoneum

revealed a well rounded, smooth, soft tissue mass 6 by 5 by 4 cm., located in the left leaf of the diaphragm, moving synchronously with the diaphragm. Preoperatively a diagnosis of benign tumor (cyst vs lipoma) of the diaphragm was made (Figs 1, 2, and 3).

On Oct 13, 1953, a thoracotomy was done through the bed of the left eighth rib, and a large, paper thin, walled cyst measuring 6 by 5 by 4 cm was found anteriorly and laterally in the dome of the diaphragm. There were no adhesions present. The cyst was easily removed by sharp dissection as the diaphragmatic musculature was completely absent in this area. On removal of the cyst a defect was left in the diaphragm, being covered inferiorly by diaphragmatic peritoneum only. This defect was closed with interrupted mattress sutures of 00 black silk. The chest was closed without drainage. The patient had an uneventful recovery and has been asymptomatic to date.

The cyst was filled with clear, straw colored fluid. The wall was lined by a single layer of flat mesothelium and strands of fibrous tissue (Figs 4, 5, 6, and 7).

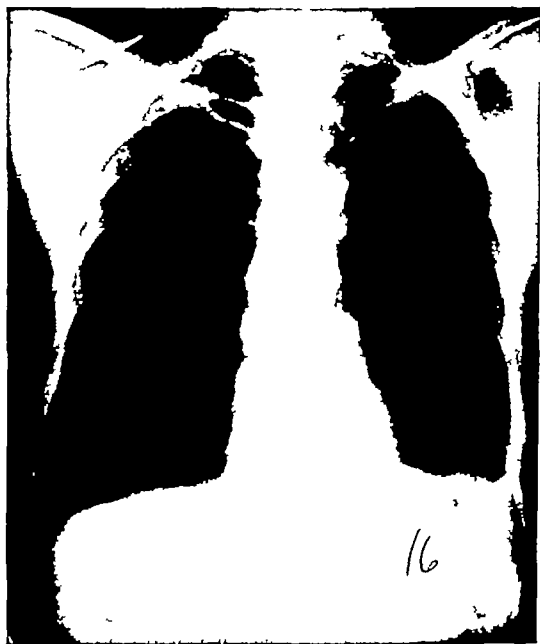


Fig 7—Postoperative film of chest (February 1954)

DISCUSSION

Primary tumors of the diaphragm are rare. Up to the present time, there were only eight other primary cysts of the diaphragm recorded. Many other mesothelial cysts, i.e., "celomic cysts," have been recorded. We believe with Cruickshank and Cruickshank³ that these cysts are comparable and form one of a series of cysts arising in different anatomical positions from various parts of the mesothelial lining of the celomic cavities. Celomic cysts are to be differentiated from cysts arising by different embryologic processes from different embryologic structures, i.e., bronchogenic, mesenteric, lymphatic, etc.

There are no true diagnostic signs and symptoms. Many are asymptomatic. The symptoms are varied but tend to follow a pattern of diaphragmatic irritation. Some symptoms, as referred pain to the shoulder, neck, or back, are due

to involvement of branches of the phrenic nerve. These tumors may cause cough, dyspnea and hemoptysis by compression of the lower lobe of the lung and effusion by irritation. Gastrointestinal symptoms have been encountered. Depending upon their location cardiac symptoms may be present. Samson and Childress¹ report clubbing of the fingers and cardiac symptoms due to the pressure of what amounted to a pulmonary arteriovenous fistula in a malignant tumor of the diaphragm. In diagnosis a loculated empyema may be troublesome.

SUMMARY

1 Primary tumors of the diaphragm are rare. Benign tumors are slightly more prevalent than malignant tumors.

2 Diaphragmatic cysts may be asymptomatic. When symptoms are present, they are due to size and position of the tumor. The usual symptoms are cough, dyspnea and occasional hemoptysis with referred pain to the shoulders and back.

3 Diagnosis is made by roentgenograms and fluoroscopy utilizing pneumoperitoneum and pneumothorax when necessary.

4 Treatment is excision.

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RADICAL EXCISION OF MALIGNANT CHEST WALL TUMORS

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PRIMARY or secondary malignant neoplasms occasionally involve the deep structures of the chest wall. Sarcomas of various types account for most of the primary lesions. Recurrences following radical mastectomy for carcinoma of the breast comprise the majority of secondary tumors, and direct extension from the lung is not infrequent. Treatment of these lesions until recently has been unsatisfactory, and even today many surgeons are reluctant to undertake radical excision, and not infrequently rely upon palliative measures. Any attempt at surgical cure should consist of wide radical extirpation.

When resection of the bony chest wall is accomplished and the defect simply covered by soft tissue, instability is certain to occur. Extensive paradoxical motion, especially in the presence of a mobile mediastinum, can lead to serious complications in the immediate postoperative period. For this reason, many surgeons in the past have avoided surgery in this location or compromised the extent of excision required.

Numerous attempts to stabilize large defects, using autogenous grafts of bone, cartilage, and fascia have been described. These methods have been quite successful in some hands, but, as a general rule, are time consuming. Essentially, a second operation is performed on a patient who has already undergone extensive surgery.

In 1947, one of us (J M B)¹ attempted prosthetic repair of large chest wall defects using tantalum plate which was molded and sutured to the surrounding bony structures with wire. Good stability was obtained in the immediate postoperative period, but the plates were poorly tolerated and had to be removed within a few weeks. Since permanent stability is more desirable in these cases, plates were abandoned in favor of tantalum mesh.

We² recently published a report on the use of mesh prostheses in two cases. The material was kindly tolerated by the tissues and seemed to give satisfactory long-term support. Other writers appear to share these opinions. Tearing of implanted mesh has been observed by some authors. The acute stresses of constant chest motion have a wearing effect on mesh fibers, and fracture may occur in time. Although tearing has not been detected in any of our cases, the implantation technique has been modified because of these reports. Originally, mesh was sutured to bone and chest wall fascia about the edges of the defect. Two layers of mesh are now employed, and since fixation of mesh to moving bone may predispose to tearing, the material is allowed to overlap the edges of

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the defect about 1 inch and is tacked snugly to fascia with interrupted cotton sutures. It seems reasonable to assume that fascia exerts some cushioning effect when transmitting the stresses of chest wall motion. Stability obtained with this technique has been excellent. Stainless steel mesh with which we have had no experience is claimed to have even less tendency to fracture than tantalum and certainly seems deserving of further trial.

Once the mesh has been secured over the defect, it must be covered by soft tissue. Simple undermining of wound edges, sliding flaps and pedicle flaps have all been used in different situations. When dealing with anterior chest wall defects mobilization of the entire opposite breast and underlying pectoral muscle provides a large and convenient donor flap. This procedure is technically quite simple and has provided very satisfactory coverage each time it has been used. There has been no difficulty with wound healing in any of our extensive resections. All except one in which there was a partial breakdown of a sliding flap have healed per primam and in this instance despite the fact that underlying mesh was exposed granulation filled the area rapidly and the wound healed uneventfully.

The purpose of the present paper is to present ten consecutive cases of chest wall malignancy treated by radical excision and prosthetic repair. Once the diagnosis of chest wall sarcoma or carcinoma has been established, curability of the lesion should be considered. A careful clinical and roentgenographic search for metastases is in order if one is dealing with a primary lesion and detectable spread elsewhere must be ruled out in secondary tumors.

The procedure should be limited to cases considered curable and has not been recommended to any patient as a palliative measure. In those cases where cure was not achieved however worthwhile palliation was obtained for considerable periods of time.

CASE REPORTS

Primary Carcinoma of Lungs—

CASE 1—W. M., a 72 year old white man, was admitted to the Rhode Island Hospital on April 29, 1948 because of symptoms and x-ray findings consistent with a malignant neoplasm of the lower lobe of the left lung. There was in addition x-ray evidence of tumor extension to the adjacent seventh rib.

On May 6, 1948, radical en bloc excision included left lower lobectomy, large segments of the fifth, sixth, seventh, and eighth ribs with the overlying chest wall soft tissues. The chest wall defect was closed by suturing a tantalum plate to the surrounding ribs with wire. Pathology report was a large bronchiogenic carcinoma measuring 7 by 4.5 by 5 cm. with invasion of the pleura and the adjacent sixth and seventh ribs.

Postoperatively, chest wall stability was very satisfactory but there was considerable drainage from the wound. Six weeks later, because of persistent drainage and local pain, the plate was removed.

The patient experienced complete relief of symptoms and rapidly gained weight. When seen on the last follow up visit on April 9, 1954, six years after resection (Fig. 1), he stated he led an active life and felt fine. There were no clinical or x-ray signs of recurrence.

CASE 2—L. M., a 52 year old white man, entered the Rhode Island Hospital in July, 1950, because of symptoms and x-ray findings consistent with a malignant neoplasm of the upper lobe of the right lung.

In August, 1950, exploration revealed a huge tumor which was firmly attached to the third and fourth ribs. A right pneumonectomy with en bloc excision of the second, third, fourth and fifth ribs and overlying chest wall soft tissues was accomplished (Fig 2). The defect was closed by suturing tantalum mesh to the chest wall fascia with interrupted cotton. Pathology report was adenocarcinoma of the lung with peribronchial lymph node metastases.

Recovery was uneventful (Fig 3). He experienced dramatic relief of symptoms and was able to resume his work in a textile mill for four months. Weakness and weight loss then recurred and he was referred to a Veterans Hospital for further study. He died one year after surgery of recurrent disease. The chest wall was described as very stable by physicians in attendance prior to his death.



Fig 1—Case 1 Photograph of patient six years after operation

Recurrent Tumors of the Breast —

CASE 3—L S, a 54 year old white housewife, entered the Rhode Island Hospital on Dec 4, 1949. Seven years before, a left radical mastectomy had been performed for carcinoma of the breast with axillary metastases. She did well until sixteen months prior to admission when a mass appeared on the left anterior chest wall. X ray treatments were recommended by her physician. There was temporary tumor regression, but three months before admission the mass again began to grow. It became extremely painful, and she lost 45 pounds. Hormones and more x ray therapy were prescribed without benefit. She was then referred to the hospital (Fig 4).

Physical examination revealed a 4 by 8 cm firm mass which was fixed to the sternum and left clavicle. On Dec 6, 1949, en bloc excision was done which included the upper third of the sternum, wide segments of the left clavicle and upper three ribs, the underlying phrenic nerve, and all overlying soft tissues. The chest wall defect was repaired by suturing a tan

Fig 2



Fig 3

Fig 2—Case 2 Specimen of right lung with adjacent wall removed at operation
Fig 3—Case 2 Appearance of wound nine months after surgery (Reproduced by permission of The New England Journal of Medicine.)



Fig 4—Case 3 Appearance of lesion before operation



Fig 5—Case 3 Photograph of patient 4 years and 5 months after surgery illustrating the use of the opposite breast and pectoral muscle as a pedicle flap Note that nipple has been excised

talum plate to the surrounding bony structures with wire. The right breast and pectoral muscle were then mobilized and swung as a flap graft to supply soft tissue coverage. Pathology report was adenocarcinoma of the chest wall invading the sternum and left sterno-clavicular joint.

Her immediate postoperative course was uneventful. One month after surgery, however, she was readmitted to the hospital because of severe local pain and recurrent pleural effusions requiring thoracenteses. The plate was removed. There was no further effusion or pain, and the wound quickly healed.

She rapidly regained her lost weight and resumed her household duties. When seen on last follow up visit on April 8, 1954, over four years later, she was leading an active, unrestricted life and had no complaints. There were no clinical or x-ray signs of recurrent tumor. (Fig 5)

CASE 4—H. H., a 72 year old white widow entered the Rhode Island Hospital in June 1950. Ten years before, a left radical mastectomy was done for carcinoma of the breast. Two years prior to admission, a lump appeared on the left anterior chest wall. Tablets were prescribed by her physician, but the mass slowly enlarged. Three months before entry, the mass began to grow rapidly and finally became so painful that hospitalization was recommended.

Physical examination revealed a 4 cm. firm tumor which was fixed to the left second costal cartilage.

En bloc excision included the upper two thirds of the sternum, wide segments of the first three ribs bilaterally, the medial third of the left clavicle, and all overlying tissues. The mass extended deeply into the mediastinum. Segments of the upper lobe of the left lung and left phrenic nerve were removed to encompass the growth. The chest wall defect was repaired by suturing a tantalum plate to the surrounding bony structures with wire. A circumferential incision was then made about the lower two thirds of the right breast and pectoral muscle, which were swung across the chest to supply soft tissue coverage.

Pathology report was recurrent adenocarcinoma of the chest wall. Her wounds healed well and the immediate postoperative course was benign. Three weeks after surgery, however, the wound suddenly opened and bled profusely. She was readmitted to the hospital, and the plate was immediately removed.

During the next year, the patient resumed her usual activities and had no complaints. In July, 1951, chest pain recurred and a nodule was felt at the upper margin of the sternal stump. At re-exploration, numerous nodular recurrences were found in the adjacent ribs, sternum, and underlying mediastinum. All visible tumor was excised, but it was felt that further radical surgery was hopeless. The soft tissues were closed and no attempt was made to reinforce the chest wall. The pathology report was recurrent adenocarcinoma. Recovery was uneventful, and x-ray therapy was recommended on discharge.

Surprisingly enough, she had complete relief of chest pain following surgery. She was able again to resume her usual activities and has traveled South during the past two winters.

At her last follow up check in April, 1954, she complained of pain in both hips. X-ray revealed metastases to the pelvic bones. Some relief has been obtained from testosterone, and, in spite of metastases, she at present leads a moderately active life nearly four years after her original operation.

CASE 5—M. S., a 62 year old white housewife, entered the Rhode Island Hospital on March 8, 1951 because of a tumor of the right anterior chest wall. Sixteen months previously a right radical mastectomy had been done for carcinoma of the breast. Nine months later, a lump appeared on the anterior chest wall. This was locally excised and proved to be adenocarcinoma. A week before the present admission, the patient noted another lump in the same location.

Physical examination revealed a firm, fixed, 4 cm. mass beneath the medial portion of the right clavicle.

En bloc excision included the medial half of the right clavicle, wide segments of the first three ribs on the right, the upper third of the sternum, the first three costal cartilages on the left, and all overlying soft tissues. The chest wall defect was repaired by suturing tantalum mesh to the chest wall fascia and cervical fascia above with interrupted cotton sutures. The left breast was then mobilized as in the previous cases for soft tissue coverage (Fig 6). The pathology report was recurrent adenocarcinoma of the chest wall.

The patient tolerated the surgery well. She received 500 c.c. of blood in the operating room and returned to the ward in good condition. About seven hours later, she went into sudden vascular collapse and died within a few moments.

Autopsy revealed bilateral atelectasis due to aspiration of tracheobronchial secretions. No gross or microscopic evidence of tumor could be found anywhere in the body.

These patients not uncommonly have difficulty coughing up bronchial secretions during the first day or two following surgery. This tragic and preventable death seems to emphasize the importance of the constant and meticulous care one must devote to bronchial toilet during this period.



Fig 6—Case 5. Demonstrating defect and method of mobilizing the opposite breast to repair the soft tissue defect.

CASE 6—M. M., a 61 year old white woman, entered the Rhode Island Hospital on April 26, 1951, because of an anterior chest wall mass. The lump had been present for sixteen months and had recently become painful. A right radical mastectomy had been performed seven years previous for carcinoma of the breast.

Physical examination revealed a 5 cm., firm, immovable mass to the right of the sternum in the third interspace.

En bloc excision included large segments of the third, fourth, and fifth ribs bilaterally, the interposed sternum, and all overlying soft tissues (Figs 7-9). The chest wall defect

was repaired by suturing tantalum mesh to the surrounding fascia with wire. A few wire sutures were encircled about adjacent ribs. Soft tissue closure was accomplished by rotation of the opposite breast as in the previous cases. Pathology report was recurrent adenocarcinoma of the chest wall. The patient's post-operative course was uneventful, and she was discharged ten days after surgery (Fig. 10).

During the next year, she carried on with her usual activities and felt very well. In February, 1953, however, she began complaining of weakness and weight loss. She died four months later of widespread metastatic carcinoma.



Fig. 7—Case 6. Defect following excision of lesion. (Photographic Dept. Rhode Island Hospital)

CASE 7—N. O., a 69-year-old white housewife, entered the Rhode Island Hospital on May 23, 1951, because of a lump of the right anterior chest wall of two weeks' duration. A right radical mastectomy had been performed two years earlier for carcinoma of the breast with axillary node metastases.

Physical examination revealed a firm, fixed, 4 cm. mass in the right sternoclavicular area.

Radical excision included the medial third of both clavicles, wide segments of the first and second ribs bilaterally, the upper 8 cm. of sternum, and all overlying soft tissues. The chest wall defect was closed with a double layer of tantalum. The mesh was allowed to extend 1 inch beyond the edges of the defect and was sutured to fascia with interrupted cotton. Soft tissue closure was accomplished by rotation of the opposite breast. Pathology report was recurrent adenocarcinoma of the chest wall.

Her postoperative course was uneventful and chest wall stability excellent. Unfortunately, she died at home of a myocardial infarct one month after discharge. Permission for autopsy was not obtained.

CASE 8—D. T., a 56-year-old white housewife, entered the Rhode Island Hospital on June 21, 1953. She had noticed a lump of the right anterior chest wall for over two years. It had been increasing in size slowly, and recently it had become painful. A left radical mastectomy had been performed in 1935 for carcinoma of the breast.

Fig 8



Fig 9

Fig 8—Case 6 Anterior view of excised specimen

Fig 9—Case 6 Posterior view of specimen Note tumor bulging on mediastinal surface

Physical examination revealed a large, firm, fixed mass between the second and third ribs of the right anterior chest wall.

At operation, a 12 by 6 by 4.5 cm ovoid mass was found to bulge into the pleural cavity. It involved the third rib but was not attached to the underlying structures. Radical excision included the second, third, and fourth ribs from the sternum to their paravertebral attachments, and all overlying soft tissues. The chest wall defect was repaired by suturing a double layer of tantalum mesh to chest wall fascia with interrupted sutures. Mesh was allowed to overlap the edges of the defect for about one inch (Fig 11). Soft tissue closure was accomplished easily by undermining. Pathology report was recurrent adenocarcinoma of the chest wall.

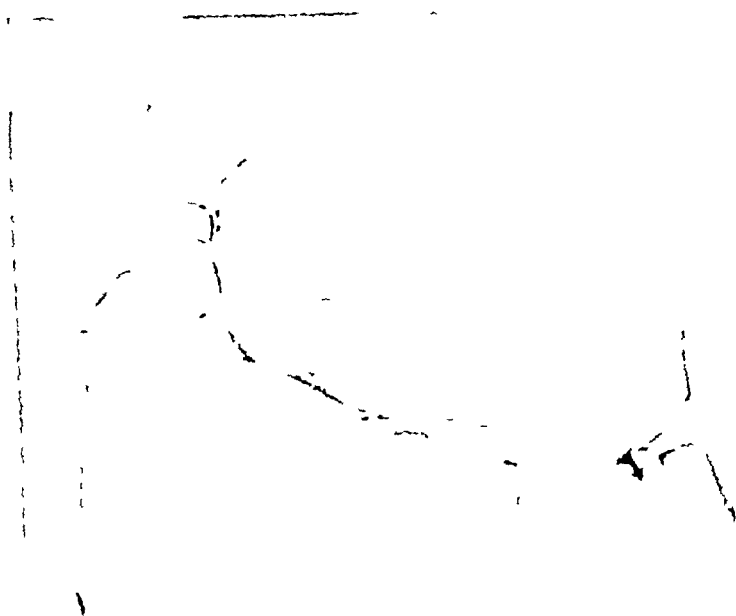


Fig 19—Case 6. Appearance of anterior chest wall ten days after operation. (Photographic Dept., Rhode Island Hospital.)

The patient's postoperative course was uneventful. Chest wall stability was excellent. She was discharged on the tenth postoperative day. When seen last on April 2, 1954, she was feeling well, had a good appetite, and was leading an active life. There were no clinical or x-ray signs of recurrence.

CASE 2—C. T., a 63-year-old white widow, entered the Rhode Island Hospital in February 1947 because of a rapidly enlarging mass of the anterior chest wall first noted six months before. X-rays revealed extensive generalized Paget's disease. In the manubrium, a 5 to 6 cm., expanding destructive lesion had eroded the sternoclavicular joints. The mass was visually obvious and very firm on palpation (Fig 12).

Radical en bloc excision included wide segments of both clavicles, the upper three ribs bilaterally, 10 cm. of upper sternum and all overlying soft tissue.

The chest wall defect was repaired by suturing a tantalum plate to the adjacent ribs with wire. Soft tissue coverage was obtained by rotation of the left breast. Pathology report was osteogenic sarcoma of the manubrium with invasion of the sternoclavicular joints.



Fig 11—Case 8 Showing double layer of tantalum mesh in place

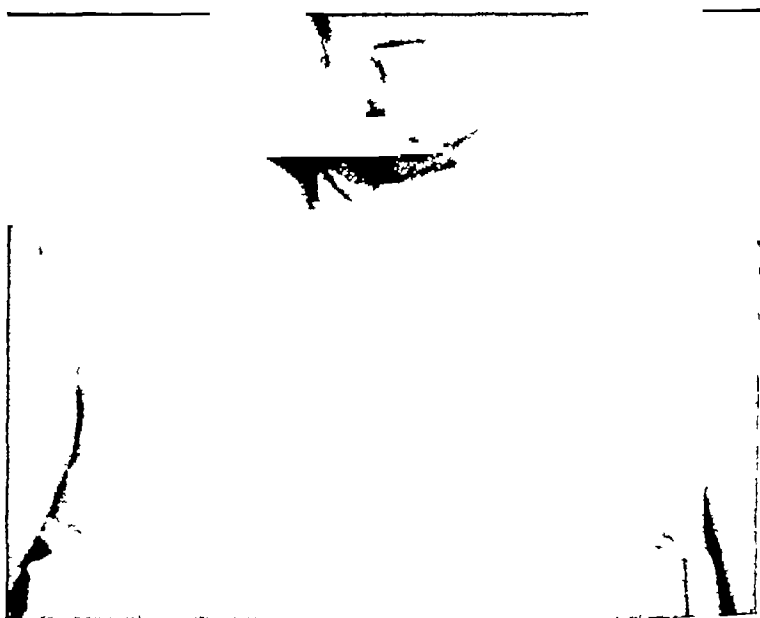


Fig 12—Case 9 Osteogenic sarcoma of sternum (From Beardsley J Thoracic Surg March 1950) Patient was living without recurrence 7 years and 2 months after radical excision

The patient's postoperative course was complicated by considerable local pain and drainage from the wound. Three months after surgery, she was readmitted to the hospital because of persistent pain and drainage. The plate was removed. She quickly regained her sense of well being and the wound healed uneventfully.

When last seen in April, 1954, over seven years post resection, she was leading an active life and had no complaints. There were no clinical or x ray signs of recurrence.

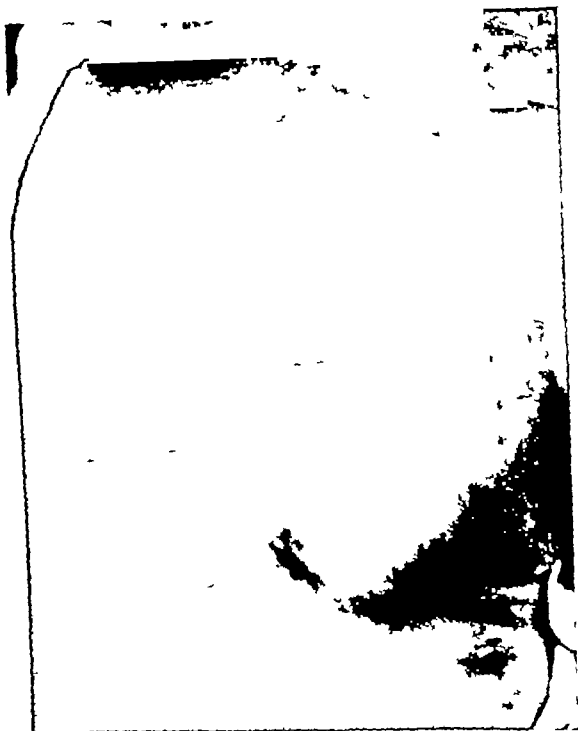


Fig 13—Case 10 Rhabdomyosarcoma of right posterior chest wall

CASE 10—M. G., a 77 year old white widow, entered the Rhode Island Hospital in August, 1950. She complained of an extremely painful grapefruit sized mass of the right posterior chest wall (Fig 13). The tumor had been present for six months, was increasing in size rapidly, and she had lost 30 pounds.

Radical excision included large segments of the seventh, eighth, ninth, tenth, and eleventh ribs, some underlying pleura, and all overlying soft tissues (Fig 14). The chest wall defect was repaired by suturing tantalum mesh to chest wall fascia with interrupted cotton sutures. A few sutures encircled the sixth rib above and the twelfth rib below. Soft tissue coverage was accomplished by utilizing a sliding flap.

Pathology report was rhabdomyosarcoma of chest wall.

The patient was a hypertensive, elderly, debilitated woman. Her convalescence was prolonged and complicated by a partial breakdown of the sliding graft. Despite the fact that underlying mesh was exposed, the area granulated in and healed solidly. She was discharged on the sixty-first hospital day (Fig 15).

During the next two years, she did well. Her weight was maintained, and she was able to perform her usual chores about the house. In August, 1952, however, she began complain

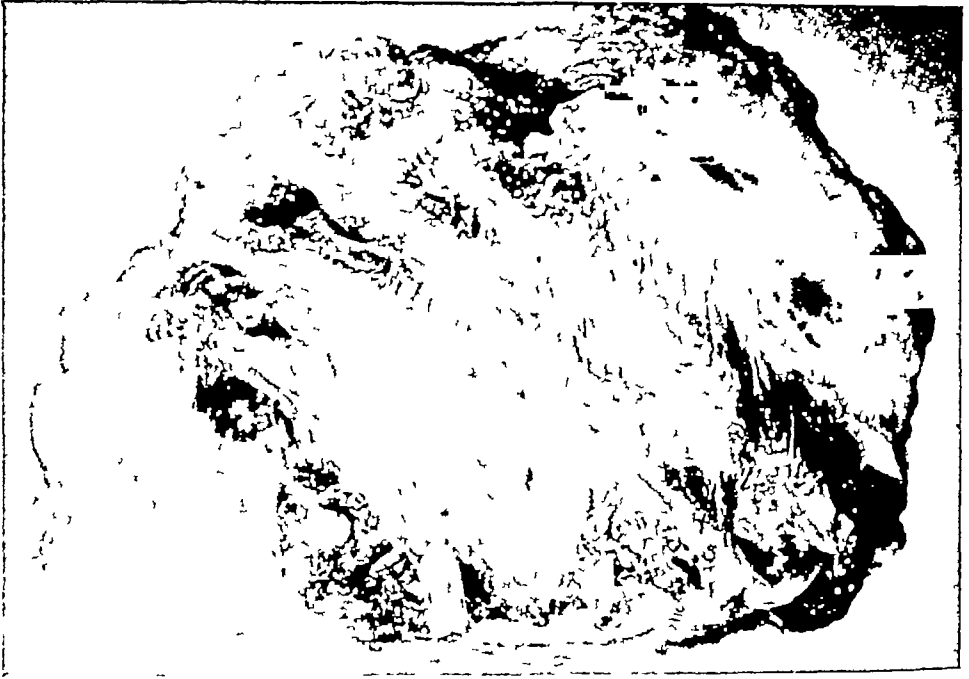


Fig 14—Case 10 Specimen showing amount of chest wall resected

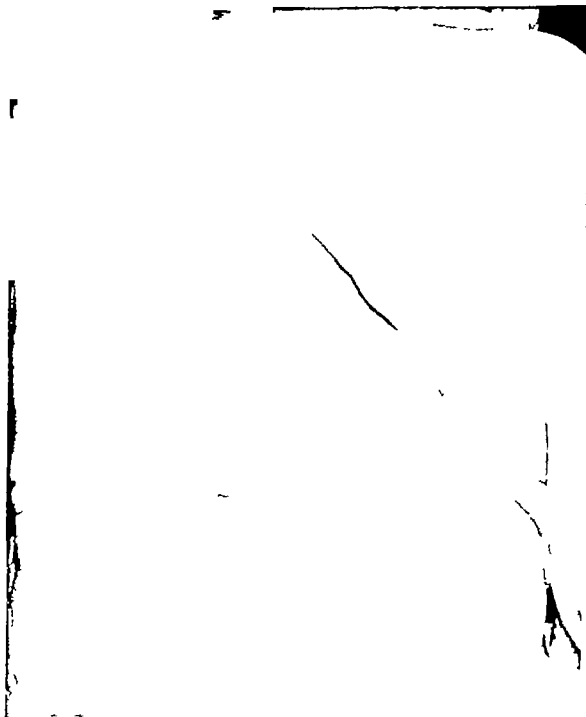


Fig 15—Case 10 Patient nine months after surgery. (Reproduced by permission of The New England Journal of Medicine.)

ing of weakness and back pain. Shortly after this she was sent to a nursing home for care. While there, a large recurrent tumor mass appeared in the presacral area. She ran a progressively downhill course and died in March, 1953. Permission for autopsy was not obtained.

DISCUSSION

Primary carcinoma of the lung which invades adjacent chest wall structures frequently arises in peripheral lung tissues. Hilal encroachment by these growths is less likely than with centrally located lesions, and hence pneumonectomy or lobectomy is generally not technically difficult. Invasion in our two cases was confined to bone and periosteum. Resection of chest wall was limited to rib and intercostal muscles. The overlying soft tissues were not disturbed, and local recurrence did not occur in either case. The chest wall resection adds little time to the procedure and none to the convalescence. In the absence of detectable metastases elsewhere, a reasonable salvage rate should be expected.

Fixation of breast cancer to chest wall has long been considered a sign of inoperability and because of the prevalently taught dogma many of the above cases were sent to the hospital for terminal disposition. The growths had been observed by the patients themselves an average of fourteen months before referral. Usually a wide circular incision is made about these tumors including a liberal margin of normal skin. As the incision is extended down through all layers of chest wall, carefully isolation and ligation of the intercostal and internal mammary vessels will prevent troublesome hemorrhage. Of six recurrent breast cancers so treated, one is alive and apparently free of disease more than four years after resection. Another has no evidence of recurrence ten months after surgery and in a third case, there was no gross or microscopic evidence of tumor at autopsy. A fourth patient unfortunately suffered a fatal myocardial infarct shortly after leaving the hospital. Definite recurrence has been observed in only two cases, and one of these obtained palliation of severe chest pain for over three years. Perhaps earlier referral will increase the salvage rate in these cases. Primary malignant tumors occurring on the chest wall are treated by similar wide circular excision. Since these growths are physically obvious and detected at an early stage by the patient himself, cures should be expected in a favorable percentage.

A series of ten cases is, of course, much too small to be of any clinical significance. These cases are infrequently encountered, however, and follow-up reports on large series of surgically treated chest wall malignancies are rarely seen.

Surgery for these tumors has been criticized as mutilating, dangerous, and hopeless. Actually, with prosthetic repair, physical deformity is not marked, and there should be no functional incapacity. One preventable death in ten cases can hardly be used as an argument against the safety of the procedure. All wounds are closed tightly without drainage, and healing per primam has been the rule. One elderly, debilitated woman had a partial breakdown of a graft and a protracted hospital course. Most other patients left the hospital by the tenth postoperative day.

One patient is alive seven years after resection of a large osteogenic sarcoma of the sternum, another six years after resection of an advanced epidermoid

carcinoma of the lung invading the chest wall, and a third four years after excision of a recurrent breast carcinoma that had infiltrated the bony cage

X-ray therapy rarely if ever leads to permanent cure of malignancies of the nature described above

Our experience with ten cases with seem to indicate that any chest wall malignancy, which after careful study appears to be self-limited, should be given the benefit of the doubt and widely excised

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TRAUMATIC HEMOMEDIASTINUM

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DESPITE the current emphasis on lesions of the mediastinum and the increased incidence of injuries involving the thorax the occurrence of mediastinal hematoma appears to have evoked little interest. It is believed that this complication is more frequent than the paucity of available reports would indicate. For this reason it is felt desirable to review present knowledge regarding hemomediastinum with particular attention to that following trauma and to describe an illustrative case.

Significant mediastinal hemorrhage presumably nontraumatic has been observed in association with numerous diseases including rupture of syphilitic¹ and arteriosclerotic^{2,3} aortic aneurysms, hemorrhage into parathyroid adenomas,⁴ uremia,⁵ typhoid fever,⁶ and "venulæ heredofamilial hemorrhagic diathesis." The case of hemomediastinum following rupture of an atheromatous aorta reported by Dupeirat and Ullmann⁷ would appear on examination of the pathologic data presented by these authors to have been due rather to dissecting aneurysm.

Reported instances of mediastinal hematoma secondary to trauma are almost equally sparse. Körner⁸ in 1916 described the case of a 30-year-old soldier who was injured by an exploding grenade and who developed a large posterior mediastinal hematoma which displaced both trachea and esophagus. Other authors^{9,10} have drawn attention to the association of hematomas of the posterior mediastinum with fractures of the thoracic vertebrae. Mediastinal hematoma following penetrating trauma has been reported by Horn¹¹ whose patient a 21-year-old man sustained a stab wound of the chest with laceration of the left internal mammary artery. The patient recovered and serial chest x-rays showed progressive absorption of the hematoma, resolution being complete within forty-one days. Zimmerman¹² has described the occurrence of acute hemomediastinum following a severe lateral compression injury of the chest. Respiration was never seriously embarrassed, conservative treatment was successful and repeated chest x-rays over a one-month period showed gradual absorption of the hematoma. In the case reported by Schmitt¹³ multiple chest injuries occurred as a result of a motoring accident, a huge mediastinal hematoma being associated with pulmonary parenchymal hemorrhage, hemothorax and rib fracture. Death occurred unexpectedly fourteen days after injury. Duvster's¹⁴ patient with mediastinal hematoma likewise presented multiple concomitant thoracic injuries. Vincent and associates¹⁵ have reported an instance of fatal hemomediastinum in a 12-year-old child.

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who fell 50 meters into a lake. Chest x-ray showed enormous enlargement of the mediastinum. Death supervened forty-eight hours after injury, and autopsy revealed a huge mediastinal hematoma, but the precise site of its origin could not be determined. Further attention has recently been directed to traumatic mediastinal hematoma by Endress,¹⁰ who has described two cases following chest injuries sustained in automobile accidents.

Most standard treatises on roentgen diagnosis accord but brief mention to traumatic mediastinal hematoma. Shanks and Kerley¹⁷ state that it may result from injury to a large vessel in the root of the neck, the blood gravitating inferiorly into the mediastinal areolar tissue. On x-ray the mediastinum is widened and may show straight vertical borders. Schwinz and associates¹⁸ mention that its roentgen differentiation from mediastinitis may be difficult. According to Ritvo,¹⁹ there is usually bilateral widening of the supracardiac shadow, and, if the blood remains confined to the mediastinal region, the margins of the shadow are straight, sharply defined, and smooth in outline.

The following case illustrates many of the clinical and roentgenologic characteristics of traumatic mediastinal hematoma.

CASE REPORT

On Dec 6, 1953, a 20 year old sailor was admitted to Framingham Union Hospital* at 4 A.M., shortly after an accident in which he fell asleep at the wheel of his car and struck a tree. He had not been rendered unconscious by the injury. When examined, the patient's chief complaint was difficulty in breathing. Pulse was 120 per minute and blood pressure was 90/70 mm Hg. There were numerous lacerations, abrasions, and contusions of the face, chest, and lower extremities. Neck veins were slightly distended, and moderate cyanosis was present over the head and neck. A right Horner's syndrome was noted. The trachea was displaced to the left. There were fullness and tenderness over the right upper anterior chest and over the medial aspect of the right clavicle, pressure on which elicited crepitus. The medial end of the right clavicle was movable, and small fragments of bone were palpable in the sternal notch. Subcutaneous emphysema was absent. Auscultation of the lungs was unremarkable except for decreased breath sounds at the right pulmonary base. The right radial pulse was as forceful as the left. Red cell count on admission was 4.56 million, and hemoglobin was 16.6 Gm per cent. Urinalysis was unremarkable.

Posteroanterior (Fig 1) and lateral x-rays of the chest showed a dense soft tissue mass involving the superior mediastinum bilaterally, predominantly on the right side. The trachea and presumably the esophagus were displaced to the left. There was a fracture of the second right rib anteriorly. The space between the medial ends of the clavicles was widened, suggesting the possibility of a fracture of the upper sternum. In the lateral projection the superior mediastinum was completely cloudy.

The patient was considered to have a hematoma of the anterosuperior mediastinum, chiefly on the right, secondary to laceration of superior mediastinal veins, possibly the thyroid vein, transverse cervical vein, or smaller tributaries of the right subclavian vein.

All external wounds were cleaned, sutured, and dressed. Parenteral penicillin was administered prophylactically. An infusion of 5 per cent dextrose in water was begun. The patient was less dyspneic when sitting semupright and was, therefore, maintained in this position. Oxygen was not necessary. Endotracheal aspiration was used to prevent the accumulation of secretions. Four hours after admission blood pressure was 100/70 mm Hg, and pulse 92 per minute. A small hemoptysis occurred at this time but was not repeated. The clinical condition was improved, and needle aspiration of the hematoma was not considered necessary.

*Service of Dr. Lee G. Kendall.



Fig 1—Posteroanterior chest x ray secured upon admission showing large mediastinal hematoma. The tracheal deviation is evident but bony injuries are not clearly reproduced



Fig 2—X-ray thirty hours after injury. There has been a striking spontaneous decrease in the size of the hematoma. The presence of right pleural fluid is suggested



Fig 3—Two days after trauma. Further improvement with return of the trachea to the midline and continued regression of the hematoma. Slight elevation of the right hemidiaphragm.



Fig 4—Elevation of the right hemidiaphragm is the only significant abnormality seen on this x ray taken sixteen days after injury. (Courtesy U S Naval Hospital Chelsea, Massachusetts.)

Chest x ray on Dec 7, 1953, approximately thirty hours after injury, showed striking regression of the mediastinal hematoma (Fig 2). There was however, evidence of thickened pleura and fluid accumulation on the right. On Dec 8, 1953, chest x ray showed further narrowing of the mediastinal mass (Fig 3) and almost complete absorption of the right sided pleural fluid. The right hemidiaphragm was slightly elevated. The trachea was almost in the midline.

The patient's subsequent course was one of progressive improvement, both clinically and roentgenologically. The Horner's syndrome regressed and dyspnea receded. On Dec 13, 1953, the patient was transferred to the U S Naval Hospital, Chelsea, Massachusetts. Pain in the right sternoclavicular region was the only outstanding complaint at this time. X rays showed slight separation of the right sternoclavicular joint, and compression and distraction of the joint produced pain. Physiotherapy, with active shoulder girdle exercise and diathermy, resulted in improvement. Chest x ray on Dec 22, 1953, was unremarkable except for elevation of the right hemidiaphragm (Fig 4). However, inspiration expiration x rays, together with fluoroscopy, showed normal diaphragmatic movement bilaterally. On Jan. 14, 1954, the patient was discharged to duty.

(Subsequent investigation of the patient's health record showed that his most recent chest x ray prior to injury had been secured on March 19, 1952, and indicated no abnormality.)

DISCUSSION

Mediastinal hematoma complicating disease is chiefly of academic interest both therapy and prognosis being those of the underlying condition. In such instances, unless catastrophic, the development of the hematoma is usually gradual, permitting adequate readjustment of cardiorespiratory dynamics. However, the clinical and roentgenologic findings may require differentiation from other causes of mediastinal enlargement, particularly those amenable to surgical or radiation therapy. In contradistinction, hemomediastinum due to trauma often presents the dual problem of acute blood loss and sudden alteration of cardiopulmonary physiology. As such, it may constitute an immediate threat to the life of the patient.

In view of the present high incidence of crushing chest injuries, especially of the so-called "steering-wheel" type, the apparently rare occurrence of traumatic hemomediastinum is surprising. It is felt however, that the lesion is actually more frequent than a survey of available reports would suggest. In many instances, for example, it may be considered a relatively unimportant component of a large complex of multivisceral injury. An additional reason is mentioned by Endress,¹⁵ who feels that since x-rays of patients with chest trauma are usually done at short distances and with Bucky technique, the mediastinum is scanned less carefully because of the expected distortion.

The source of the hemomediastinum in instances of blunt injury would appear almost invariably to be venous. In the present case, it was believed to result from laceration of smaller superior mediastinal veins such as the thyroid vein, transverse cervical vein or smaller subclavian tributaries. With fracture of ribs or sternum, the possibility of injury to intercostal or internal mammary veins must be considered. Fracture of the sternum may also produce copious bleeding from the vascular marrow.¹⁶ In penetrating injuries arterial hemorrhage may be predominant, as in Horn's¹¹ case of stab wound of the internal mammary artery.

Findings on physical examination in patients with traumatic mediastinal hematoma depend upon its extent, location, and rapidity of accumulation. There is usually obvious outward evidence of injury to the bony thorax. Obstruction to venous drainage may be indicated by distended neck veins. The patient may exhibit marked dyspnea, often favoring the semierect position for relief. Shock may result from reduction of circulating blood volume, especially should the hematoma rupture into the free pleural space. A transient unilateral Horner's syndrome was noted in the present case. In short, clinical findings may be suggestive but are rarely unequivocal.

The roentgen characteristics of traumatic hemomediastinum have been briefly discussed, most authors emphasizing enlargement of the mediastinal shadow with straight, smooth, sharply defined margins. Endress¹⁶ distinguishes two possibilities. In one, the findings may be most marked on posteroanterior projection with a lateral bulging of the mediastinum being evident. A second type is most apparent in the lateral x-ray, which may demonstrate a convex soft tissue shadow in contact with the posterior surface of the sternum. The hematoma in the case herein reported was most prominent in the posteroanterior view but was also clearly visualized on the lateral x-ray.

X-ray diagnosis of traumatic mediastinal hematoma in the absence of a history or roentgen evidence of gross chest injury is necessarily tenuous. On the other hand, a history of trauma should not be permitted to exclude the possibility of an underlying mediastinal neoplasm. The tendency of traumatic mediastinal hematomas to progressive and usually rapid resolution militates against diagnostic temporizing in cases which exhibit little change.

As in the patient described, conservative treatment of traumatic mediastinal hematoma is usually successful. However, especially in the early phase, signs of significant cardiorespiratory embarrassment must be promptly detected and actively treated. Decompression of the mediastinum, either by needle aspiration or by anterior mediastinotomy, may be required. In arterial hemorrhage, operative ligation of the bleeding vessels may be necessitated for secure hemostasis. Associated injuries and complications must, of course, be adequately managed.

SUMMARY

Hemomediastinum is a striking condition which may complicate the course of numerous diseases or may be associated with severe chest trauma. Its incidence following thoracic injury is believed to be higher than indicated by the literature. A case of traumatic mediastinal hematoma with successful conservative management is presented. An interesting clinical finding was the presence of a transient unilateral Horner's syndrome. The source of bleeding in traumatic hemomediastinum is usually venous but may be arterial if the injury is penetrating. Physical findings are dependent upon the extent, location, and rapidity of accumulation of the hematoma. X-ray characteristics are those of enlargement of the supracardiac shadow with straight, smooth, sharply defined margins. Conservative treatment is usually adequate although severe cardiorespiratory embarrassment may require mediastinal decompression.

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STUDIES ON FIBRINOLYTIC ENZYMES IN PATIENTS UNDERGOING THORACIC SURGERY

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IN RECENT years uncontrollable hemorrhage following thoracic operations has been reported.^{1,4} In these cases the bleeding was diffuse, did not originate in major open vessels, and was ascribed to afibrinogenemia due to fibrinolysis.

During the three-year period ending July 1, 1954, 342 major thoracic interventions such as pulmonary resection, thoracoplasty, and cardiac operations were performed on our service. In this series two severe hemorrhages occurred, both after pulmonary resection. In one case a ligature had slipped off the pulmonary artery, and in the other, bleeding was due to erosion of an intercostal artery.

In the present communication, studies are reported on fibrinolytic enzymes in the blood before, during, and after operation and in blood obtained from the open chest during operation in patients undergoing major thoracic surgery.

METHODS

Blood samples were obtained from the brachial vein, the pulmonary artery, the left atrium, and the open chest. No saline or wet sponges were used until the pleural blood samples had been taken in order to avoid dilution. The samples obtained with or without anticoagulant ($\frac{1}{10}$ volume of 0.1 M sodium oxalate) were centrifuged at 2,000 r.p.m. for 15 minutes. The plasma or serum was separated and kept at +4° C for a maximum of 3 hours until the performance of the tests.

The following materials were used:

Barbital saline buffer, pH 7.4: 11.0 Gm NaCl, 8.244 Gm sodium barbital and 0.924 Gm potassium oxalate dissolved in 2 L of distilled water.

Fibrinogen: a 600 mg per cent Bovine Fraction I (Armour Co.) solution in barbital saline buffer, freshly prepared. The material contained profibrinolysin.

Thrombin (Upjohn Co.) dissolved in saline barbital buffer to a strength of 50 units per milliliter.

Profibrinolysin prepared from human serum by the method of Ratnoff.⁵

Streptokinase-streptodornase—Varidase (Lederle Co.) dissolved in barbital saline buffer to a strength of 1,000 units per milliliter.

Fibrinolysin: a bovine preparation received through the courtesy of Dr. E. C. G. Loomis from Parke, Davis and Co.

Fibrin plate prepared according to Astrup and Mullertz,⁶ heated to 85° C for 45 minutes in order to destroy profibrinolysin.

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Tests for the presence of the following substances were performed

Fibrinogen to 0.5 ml test sample mixed with 0.4 ml buffer solution, 0.1 ml thrombin solution was added at 37° C and the mixture was examined for the appearance of a clot—*method A*

Fibrinolysin to a mixture of 0.5 ml test sample and 0.4 ml fibrinogen solution, 0.1 ml thrombin solution was added. The clot was observed for lysis at 37° C during a period of 48 hours—*method B*

A similar test was performed on a 1,024-times diluted sample in order to abolish antifibrinolysin activity—*method C*

In a third method 0.05 ml of the test sample was placed on a heated fibrin plate which was observed for the appearance of a halo at 37° C for a period of 48 hours—*method D*

Profibrinolysin to a mixture of 0.05 ml test sample, 0.05 ml streptokinase solution, and 0.4 ml fibrinogen solution, 0.1 ml thrombin solution was added, and the clot was observed for lysis at 37° C, during a period of 48 hours—*method E*

Profibrinolysin activator (fibrinolysokinase) 0.1 ml of a mixture containing equal parts of test sample and profibrinolysin solution was placed upon a heated fibrin plate which was kept at 37° C. The appearance of a halo within 48 hours indicated the presence of activator—*method F*

Antifibrinolysin titrated according to Ratnoff—*method G*

RESULTS

In the 11 cases studied, the operative and postoperative courses were uneventful, and no undue bleeding was observed. The results of the laboratory studies in these 11 cases are presented in Table I.

In all blood samples obtained from the brachial vein, the pulmonary artery, and the left atrium, adequate clots appeared within a few seconds after the addition of thrombin. In none of these samples could fibrinolytic activity be demonstrated by the different methods used. In all samples examined, profibrinolysin was present and fibrinolysokinase was absent. Antifibrinolysin titers in the blood samples from the peripheral and intrathoracic vessels did not show marked changes during operation.

All blood samples obtained from the open chest either contained clots or clotted in the test tube. In none of these samples was fibrinolysin or fibrinolysokinase demonstrated, but all contained profibrinolysin. Antifibrinolysin titers in 9 of the 10 examined pleural blood samples were markedly lower than in the samples obtained simultaneously from the peripheral or intrathoracic blood vessels. Only in one sample (Case 7), which was removed from the open chest before it clotted, was the serum antifibrinolysin titer increased as compared with the titer in the peripheral blood.

COMMENT

Lung tissue is known to be rich in thromboplastin⁸ and tissue fibrinolysokinase.⁹ Afibrinogenemia or hypofibrinogenemia reported to occur in thoracic surgery might be explained by one of the following mechanisms (1) intra-

TABLE I STUDIES OF FIBRINOLYTIC ENZYMES IN BLOOD OBTAINED FROM THE PERIPHERAL AND LESSER CIRCULATION AND FROM THE OPEN CHEST IN THORACIC OPERATIONS

	ORIGIN OF BLOOD SAMPLES	PRESENCE OF CLOTS	FIBRINOGEN METHOD A	FIBRINOLYSIS METHOD			IROFIBRINOLYSIS METHOD F	FIBRINOLYSIS METHOD F	ANTIFIBRINOLYSIS TITER, CASE						
				B	C	D			1	2	3	4	5	6	7
LUNG SURGERY 7 Cases	<i>Open Chest</i>														
	Before lung resection	(7)+	(6)-, (1)+*	(7)-	(7)-	(4)-	(7)+	(7)-	15	30	50	100	100	96	800
	After lung resection	(7)+	(6)-, (1)+*	(7)-	(7)-	(4)-	(7)+	(7)-							
	<i>Brachial Vein</i>														
	Before operation		(7)+	(7)-		(5)-	(7)+	(5)-	280	150	300	500	600	360	800
	During operation		(3)+	(3)-		(3)-	(3)+	(2)-	300						800
HEART SURGERY 4 Cases	At end of operation		(4)+	(4)-			(4)+	(4)-	300		300	150		360	800
	Twenty four hours after operation		(2)+	(2)-			(2)+	(2)-	300	150					
	<i>Open Chest</i>														
	Before intracardiac procedure	(3)+	(3)-	(3)-	(3)-		(3)+	(3)-							
	After intracardiac procedure	(3)+	(3)-	(3)-	(3)-		(3)+	(3)-							
	<i>Pulmonary Artery</i>														
	During operation		(4)+	(4)-			(4)+	(4)-	600		300				
	<i>Left Atrium</i>														
	During operation		(4)+	(4)-			(4)+	(4)-	600		300				
	<i>Brachial Vein</i>														
	Before operation		(3)+	(3)-			(3)+	(3)-	550	350	300				
	At end of operation		(2)+	(2)-			(2)+	(2)-	450	120	225				

Numbers between parentheses indicate the number of cases examined

*Blood obtained from thorax before clotting clotted in test tube

vascular clotting by thromboplastin released from the lung tissue into the circulation, (2) activation of plasma profibrinolysin to fibrinolysin by tissue kinase from the lungs⁹, and (3) release of active fibrinolysin from the tissues into the circulating blood¹⁰

In our 11 cases, severe hypofibrinogenemia did not occur. Also no active fibrinolysin or fibrinolysin activator could be demonstrated in the blood. Even blood samples obtained from the open chest which had been in contact with traumatized tissue, did not show fibrinolytic activity. A remarkable as yet unexplained finding is the low antifibrinolysin titer in the pleural blood samples as compared to those obtained by puncture of blood vessels. The decrease in antifibrinolysin titer in these samples did not seem to have clinical significance since fibrinolytic activity or undue bleeding did not ensue.

It follows, therefore, that the appearance of fibrinolytic activity in the peripheral and the pulmonary circulation in thoracic surgery is not a common occurrence. This is in consonance with the absence of undue diffuse bleeding in our surgical material. When hypofibrinogenemia does occur in thoracic operations, an additional so far unknown mechanism must be involved.

SUMMARY

In patients undergoing lung resection or intracardiac surgery and who had no undue diffuse bleeding or marked hypofibrinogenemia fibrinolysin and fibrinolysokinase could not be demonstrated in blood from the peripheral veins, the pulmonary artery, and the left atrium, while profibrinolysin was present.

Blood obtained from the open chest contained clots and did not show fibrinolysin or fibrinolysokinase activity but had markedly lowered antifibrinolysin titer.

Afibrinogenemia appears to be uncommon in thoracic surgical patients and the mechanism of its occurrence is not understood.

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USE OF THE LITTLE FINGER FOR MITRAL COMMISSUROTOMY

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IN THE MAJORITY of patients with uncomplicated mitral stenosis the operation of mitral commissurotomy is a technically straightforward and effective procedure, as is attested to by the low operative risk and favorable postoperative improvements reported for these patients by many surgeons in widely differing geographic areas

In the occasional patient in whom the left auricular appendage is too small to admit the forefinger, however the morbidity and mortality of the operation are significantly higher. In such a case one must either forego exploring the heart (which hardly seems an adequate solution to the problem) or approach the diseased valve through the left atrial wall proper or through one of the left pulmonary veins. In our own experience, each of these latter two approaches has been associated with significantly greater and occasionally formidable technical difficulties of the nature which have been described by Bailey¹ and which account for the greater risk and less favorable results when these approaches are utilized by necessity.

Recently we have encountered two such patients in whom the auricular appendages would not come close to admitting the operator's forefinger, but were of sufficient size to admit the fifth finger. In both cases the commissurotomy was accomplished with surprising ease and a gratifying effectiveness—so much so that we believe it of value to call to the attention of other surgeons the value of this simple maneuver.

At first thought one might consider the use of the little finger like sending a "boy to do a man's job", however when one considers the fact that the fifth finger may be rigidly fixed in a semiflexed position and that the whole hand and arm may then be utilized to lift the fifth finger one sees that surprising leverage can be exerted to split the anterior commissure. Moreover, the shorter length of the fifth finger as compared to the forefinger is not as great a handicap as one might surmise because inserting the shorter fifth finger through the left auricular appendage actually puts one in a position appreciably closer to the mitral valve than can be obtained by introducing the longer forefinger through the more distant superior pulmonary vein. This observation is dependent upon the fact that in the average individual the

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difference in length between the forefinger and the fifth finger is less than 1.5 cm, whereas the left auricular appendage is 3 to 5 cm or more closer to the mitral valve as compared to the left superior pulmonary vein

Also, if it is deemed necessary, one may further decrease the diameter of the finger and take advantage of the sharper fingernail edge by removing the rubber glove from this finger

The following two case reports illustrate the application of this technique for commissuotomy

A. B. (U.H. No. 871612), a 47 year old man, was admitted to the University Hospitals on June 7, 1954. This patient was first known to have cardiac difficulty six years prior to admission when a routine chest x ray disclosed an enlarged heart. He had no history suggestive of rheumatic fever. Shortly thereafter he was hospitalized for decompensation and treated with digitals, diuretics, and low salt diet. He had continued daily digitals and intermittent diuretics with no further bouts of decompensation. For the two years preceding this admission he has had progressive exertional dyspnea such that he was unable to be gainfully employed.

Physical examination revealed an enlarged heart, rumbling diastolic murmur at the apex, accentuated P, and auricular fibrillation. He was well compensated without evidence of pulmonary or peripheral edema or an enlarged liver.

Surgery was undertaken on June 10, 1954, utilizing a left lateral approach through the bed of the fourth rib. A diastolic thrill was palpable at the upper border of the left ventricle. After opening the pericardium, the left auricular appendage was noted to be very small, and it was doubtful that it could be used to enter the left atrium. A purse string suture was placed about its base, however, and the appendage was opened and an attempt to insert the forefinger was made without success.

We were about to tie off the appendage and approach via the left atrial wall proper or pulmonary vein when it was suggested that an attempt be made to insert the little finger. This was subsequently done, and exploration disclosed a stenotic valve with an orifice of 5 mm. The anterior commissures were widely split with ease, using the fifth finger. The result was most gratifying, and the patient had an uneventful postoperative convalescence.

I. H. (U.H. No. 870863), a 48 year old white woman, was admitted to the University Hospitals on June 2, 1954, with a history of rheumatic fever at 14 years of age and with the onset of exertional dyspnea 15 to 20 years prior to this admission. She had first become decompensated nine years previously and had had intermittent bouts of failure with ascites, hepatomegaly, and persistent auricular fibrillation despite digitals, low salt diet, and the occasional use of mercurial diuretics.

Physical examination revealed evidence of cardiac enlargement, mid diastolic rumbling murmur at the apex, auricular fibrillation, enlarged liver, 2+ pitting edema of both ankles, and occasional râles at both lung bases.

Electrocardiogram revealed right axis deviation and atrial fibrillation. Cardiac fluoroscopy showed right ventricular and left atrial enlargement.

With the use of mercurial diuretics she subsequently lost 10 pounds of weight. The basilar râles and ankle edema disappeared, and she was taken to the operating room for mitral valve surgery on June 18, 1954. A diastolic thrill was palpable over the upper border of the left ventricle and a systolic thrust over the left atrium. The auricular appendage was small, and again it appeared doubtful whether it would accept the forefinger. Moreover, an attempt to enter the atrium by means of the forefinger through this appendage was unsuccessful even with the glove removed from the finger. In addition there was noted an organized thrombus on the adjacent left auricular wall. Because of the previous experience in the above mentioned case, the fifth finger was utilized, with the glove over this finger cut off. This finger

entered the atrial chamber without difficulty. Exploration disclosed a thick stenotic valve with a slight amount of insufficiency posteriorly. The considerable degree of stenosis present at the anterior commissures was easily split open and without any evidence of increased insufficiency. This patient also has had an uneventful postoperative convalescence.

DISCUSSION

It is, perhaps, obvious that one may still encounter an occasional patient in whom the left auricular appendage may not admit even the little finger. In such patients one must be prepared to utilize one of the other alternatives that have been mentioned. It is our belief, however, that the use of the fifth finger in patients with very small left auricular appendages will make the need for these more hazardous approaches infrequent.

SUMMARY

One of the problems encountered in mitral valve surgery is the occasional patient with a very small left auricular appendage. In certain of these patients the use of the little finger (with or without the glove removed) introduced through the left auricular appendage allows one to perform mitral commissurotomy with greater ease and safety than an approach through either the left atrial wall or pulmonary veins.

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SUBVALVULAR MITRAL STENOSIS—ITS RECOGNITION AND TREATMENT

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IN THE performance of mitral commissurotomy for mitral stenosis, it has become more and more apparent to us that, in order to relieve the stenosis as completely as possible and also to avoid the production of mitral insufficiency, one must be extremely cognizant of the subvalvular chordopapillary supporting structures. We have elected to use the term "subvalvular stenosis" to indicate the presence of any pathologic alteration on the ventricular side of the mitral leaflets which impedes blood flow through the atrioventricular channel. This condition is caused by fusion of the papillary muscles and/or chordae tendineae on the ventricular side of the valve. It can occur with or without marked shortening of the chordae tendineae.

We have attempted to divide cases of subvalvular stenosis into three types for descriptive purposes and also for recognition and surgical alleviation at the time of operation.

In type one, the chordae tendineae are cross-fused and give the tactile impression of a bird-cage or netlike structure blocking the flow of blood (Fig 1, B). In this type, finger dissection alone has proved to be the most satisfactory solution in our experience. The finger pressure is applied downward in a ventricular direction to separate the fused tendinous groups, those to the mural and septal leaflets, respectively (Fig 1, C). If the chordae are not completely fused all the way to the papillary muscle, one can, at times, insert his finger between the two groups of chordae tendineae at the tip of the papillary muscle and apply pressure upward toward the atrium to get resultant splitting (Fig 1, D). If neither of these methods is satisfactory, one can use the finger to perforate the network by downward pressure and clear a passageway for atrioventricular blood flow. This may result in minor degrees of regurgitation, but it will remain minor so long as the major chordae to the leaflets are not broken.

In type two, there is chordo papillary fusion and marked shortening of the chordae tendineae, in some cases to a length of 0.5 cm. On digital examination, one can feel the nearness of the papillary muscles to the leaflets and can feel the Y shaped spreading of the chordae tendineae as they leave the tip of the muscle to their insertion into the respective leaflets (Fig 2, A).

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The distance between the papillary muscle and the fused valve edges is so small, however, that the standard guillotine knife, developed by Bailey, Glover, and O'Neill, cannot be easily engaged into the space. We have devised a guillotine knife which has been most advantageous in coping with this type of subvalvular stenosis (Fig 3). The lower end of this knife is blunt and probe-like and presents a golf-stick-like angle at its point of take off from the

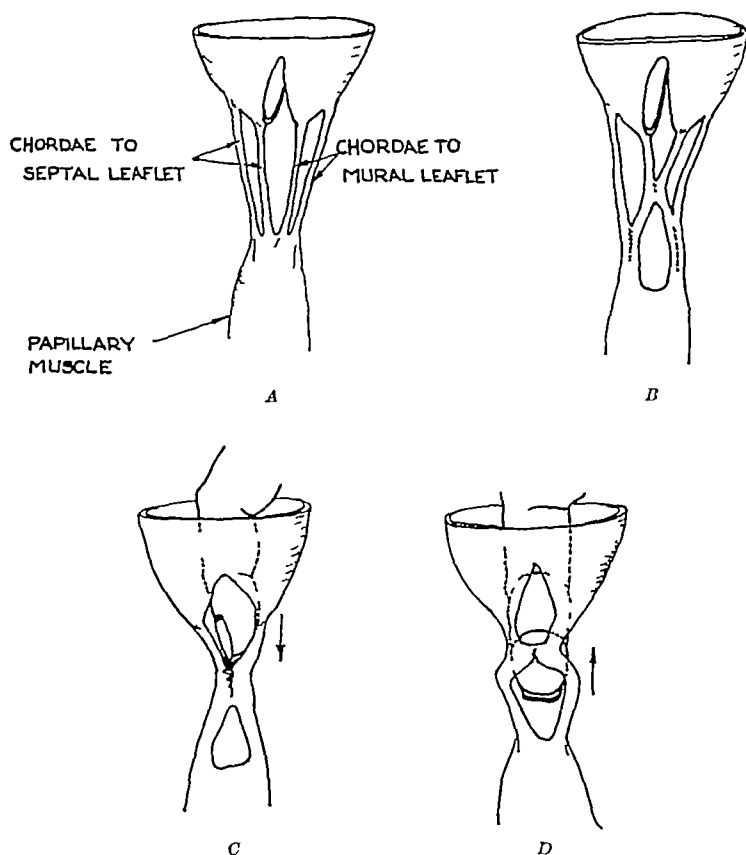


Fig 1—A Normal chordae and papillary muscle support to mitral valve. B, Cross fusion of chordae in type one subvalvular stenosis. C, Finger pressure from above to free type one stenosis. D, Finger pressure from below to free type one stenosis.

shaft of the instrument. This lower probe-like end protrudes beyond the guillotine blade when the knife is closed, but, being blunt, the closed knife has no presenting sharp edges. The blunt protruding lower end is used to seek out the passage between the papillary muscle and the mitral leaflets (Fig 2, B). After it has been engaged into this space, the instrument is then moved from side to side toward the mural leaflet and septal leaflet, respectively, to be sure that it is engaged so as to have chordae tendineae on both sides. Since there is no cutting surface on the closed instrument, several probing passes can be made with it without fear of injuring the chordae tendineae. The

engaged lower edge can be made to protrude upward against the valve so that it can be palpated by the index finger on the atrial surface of the commissure to ensure that the proper commissural alignment has been made (Fig 2, C)

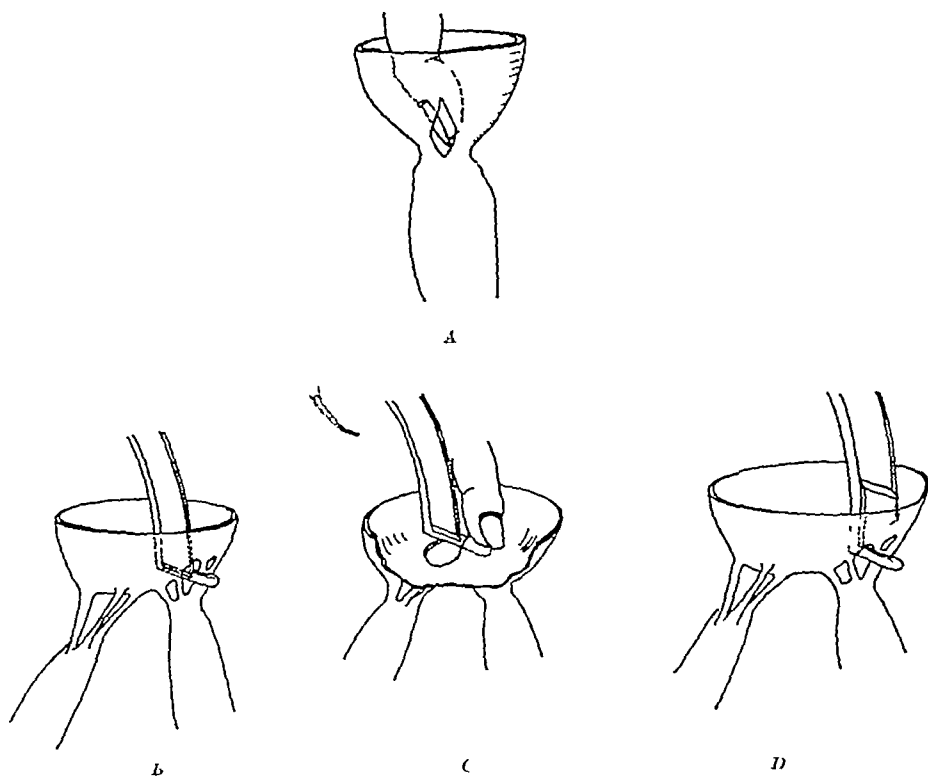


Fig 2.—A Palpation of Y-shaped fusion of thickened and shortened chordae in type two subvalvular stenosis. Papillary muscle near leaflet edges. B Probe end of closed guillotine knife engaged into small Y-shaped area beneath fused commissure. C Finger palpation on the atrial surface of the commissure to ensure proper alignment of knife. D, Opened knife is fully engaged and ready for guillotine cutting of commissure.

Next, the guillotine is opened, and the knife is engaged deeper into the commissure. The guillotine is then closed and proper commissural cutting is accomplished (Fig 2, D). It should be noted that the guillotine blade of this instrument is slightly narrower than that of the standard guillotine knife so as to ensure a smaller cut. In many instances, one small cut is all that will be necessary as the remaining commissural fusion may be split by digital pressure against the apex of the incision. If further cutting is necessary, it is better to palpate the result of the previous cut and be certain of the anatomy of the remaining stenosis before again engaging the knife to carry out opening of the commissure all the way to the atrioventricular ring. The multiple cut method is of the most importance when there is an exaggerated elliptical shape to the fused commissure. With multiple small cuts, the natural curve of the line of

valve closure can be followed safely to the atrioventricular ring, whereas a single large cut might extend peripherally into one of the leaflets to produce resultant regurgitation

After the commissure has been opened, digital pressure downward into the fork of the Y-shaped chordal attachment to the papillary muscle will often result in papillary muscle splitting which will result in greater freedom of the leaflet edges to swing widely apart during diastole. If the papillary muscle does not respond to digital splitting, a Papillotome may be used to incise the muscle. This instrument and its application will be described in the treatment of the type three stenosis

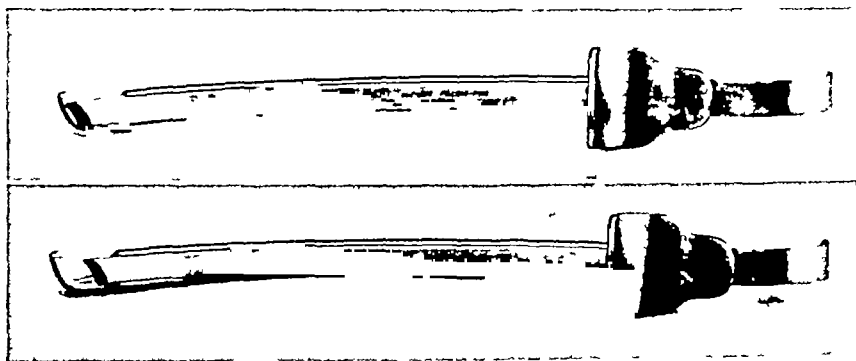


Fig 3—Guillotine knife with blunt golfstick angled lower end (Mfg by George P Pilling and Son Philadelphia Pa.)

In type three, there is leaflet papillary fusion and marked shortening of the chordae tendineae to the point that the latter are not actually identifiable as such to the palpating finger. There is actually fusion of the papillary muscles to the edges of the mitral leaflets (Fig 4, A). This type of valve is recognized when one passes his finger through the mitral orifice and extends it out beneath the commissure on the ventricular side of the valve. One then feels a thickened, fleshy bar present, but no identification of chordae tendineae can be made. A slight variation of this is the palpation of the papillary muscle mass, fusing to the mural leaflet edge, and the absence of any palpable chordae tendineae or papillary muscle to the septal leaflet edge. In this condition, the chordae from the septal leaflet are agglutinated to the ventricular surface of the fused commissure on their course to the papillary muscle.

It has been our experience that the standard guillotine knife has not been entirely satisfactory in relieving this type of subvalvular stenosis. Since the hook on the knife points back up the shaft of the instrument, it cannot be easily engaged into the papillary muscle mass so as to ensure division of this mass along with the fused commissure. It is of prime importance that the papillary muscle mass be so divided as to ensure maintenance of support to both mitral leaflets.

We have devised a guillotine knife, the "Papillotome" (Fig 5), in which the lower end has been made straight instead of containing a hook. Also, this lower end has been made to angulate from the instrument, in "golfstick" fashion so its sharp lancet point can be plunged into the papillary muscle mass perpendicular to its presenting surface. This guillotine knife, when closed,

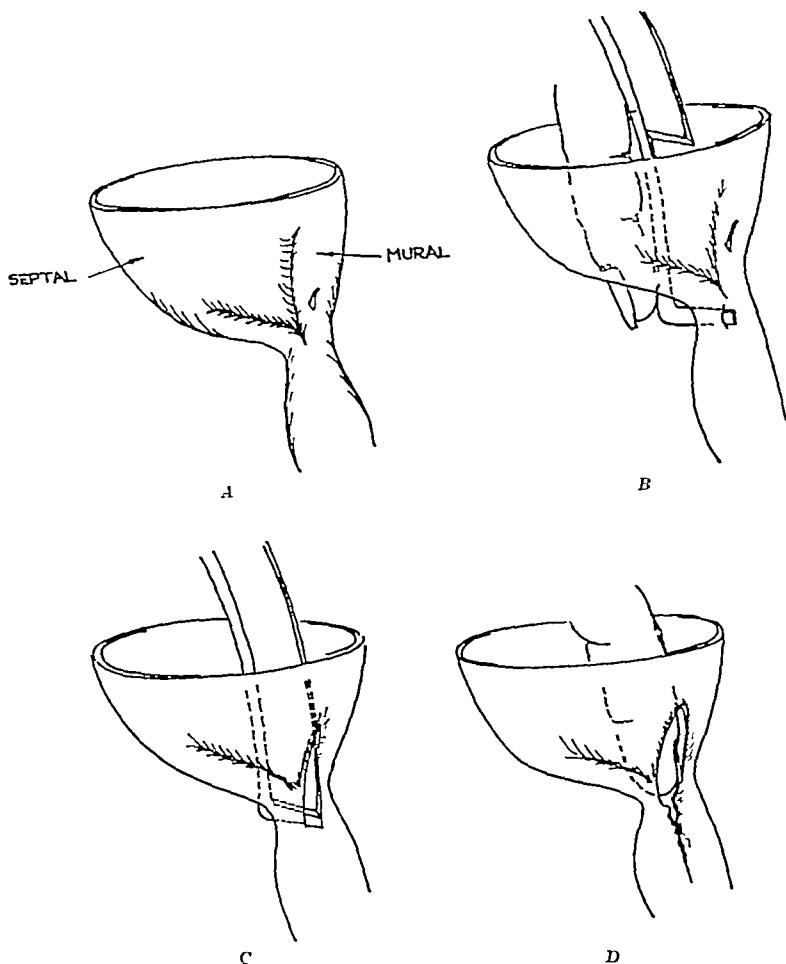


Fig 4—A Type three subvalvular stenosis where chordae are so short and thick that they cannot be definitely identified. To the palpating finger the papillary muscles seem to attach directly to the leaflet. B The lancet end of the Papillotome is inserted perpendicularly into the central area of the papillary muscle mass. C Guillotine cutting starts at the commissure on the atrial surface and extends down into the papillary muscle to the original point of Papillotome insertion. D Digital pressure to produce further splitting of the papillary muscle.

presents no cutting edges so that it can be safely introduced into and withdrawn from the heart. After the knife has been introduced into the left atrium, along the right index finger, the guillotine is opened and the lancet is inserted into the papillary muscle mass (Fig 4, B). The guillotine is then closed, and

the cutting action starts on the atrial surface of the fused commissure and extends on down into the center of the papillary muscle mass to the point where the lancet was originally engaged (Fig 4, *C*) By this method the support to each leaflet is maintained by the respective half of the divided muscle Digital pressure downward into the apex of the incision into the papillary muscle will usually result in further splitting which allows the mitral leaflet edges to fall widely apart during diastole (Fig 4, *D*) If the digital pressure is not adequate because of dense fibrosis, another cut with the knife may be necessary

DISCUSSION

The first important aspect of subvalvular stenosis is the recognition of its presence at the time of mitral commissurotomy This recognition is greatly enhanced in our experience since we have adopted the bare-finger technique The increased tactile sense of bare finger over gloved finger can be appreciated only after it has been employed in an operative case If the subvalvular stenosis is not recognized, the chordae tendineae and papillary muscles can easily be cut at the time the fused commissure is being incised

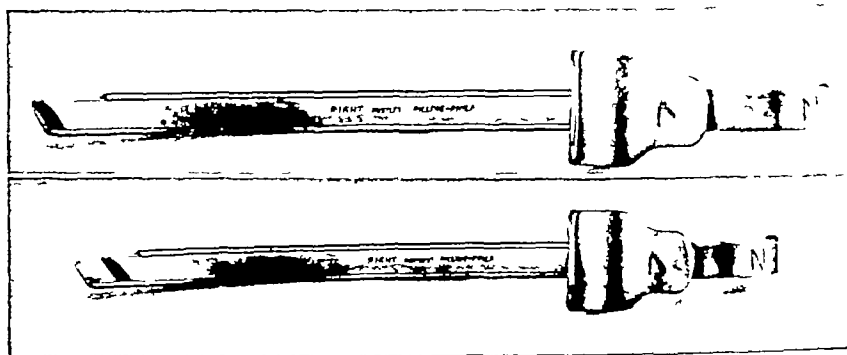


Fig 5—Papillotome (Mfg by George P Pilling and Son Philadelphia Pa.)

Such cutting of these subvalvular structures removes the support to the valve edges and invariably produces regurgitation This might well result in an immediate or delayed fatality or, at best, a poor clinical result

The second important aspect to be considered is the freeing up of the subvalvular stenosis to allow a maximum of atrioventricular blood flow Separation of fused chordae and of fused papillary muscles is essential to allow the leaflet edges to fall widely apart during diastole In the type three stenosis described, even though the papillary muscle is deeply split or incised as suggested, there is still some impedance to atrioventricular blood flow as the blood has to spill over the bulky papillary muscle mass, whereas in a normal heart, it would have only to course around the fine and delicate chordae to enter the left ventricular chamber We have no suggestion as to how this final bit of subvalvular obstruction can be overcome

SUMMARY

Subvalvular stenosis is being recognized with increasing frequency during mitral commissurotomy. Three types of subvalvular stenosis have been recognized by the ungloved palpating finger of the operator. The technique of treating each of the three types has been described. Two types of guillotine knives which have greatly augmented the successful operative technique are described for the first time.

SURGICAL TREATMENT OF MITRAL INSUFFICIENCY

EXPERIMENTAL OBSERVATIONS

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RHEUMATIC involvement of the valvular components of the mitral valve may result in mitral stenosis, a combination of stenosis and regurgitation, and what is termed "pure mitral regurgitation." Initially, there is absolute loss of valve substance by cicatricial contraction of the leaflets which prevents coaptation of the valve cusps during ventricular systole. This may or may not be associated with retraction of the valve cusps into the ventricular chamber by scarred and contracted chordae tendineae. The resultant regurgitation of blood during ventricular systole causes the left ventricle to hypertrophy and dilate. There is a delicate balance in this compensatory mechanism between the regurgitation and the ventricular hypertrophy. If there is chronic stress on the heart to increase cardiac output, sufficient ventricular hypertrophy and dilatation may result to cause dilatation of the atrioventricular ring which, in turn, causes further separation of the valve cusps. This relative loss of valve substance as a result of dilatation of the mitral ring now becomes one of the most important factors in the consideration of its surgical correction.

Examination of the insufficient valve with the intracardiac finger reveals that the regurgitant flow is entirely through the posteromedial one half of the mitral orifice. The anterior cusp of the mitral valve forms part of the aortic outflow tract during ventricular systole. The absolute, as well as relative, reduction in valve substance from the cicatricial contraction of the leaflets, with or without retraction of the leaflets into the ventricular chamber by the contracted chordae tendineae, and separation of the posteromedial commissure from dilatation of the mitral ring, gives rise to a "tear-drop" type of deformity with loss of occluding valve substance in the medioposterior one half of the orifice which allows the jet of regurgitant blood to be directed posteriorly toward the atrial septum.

Previous techniques at surgical repair of mitral insufficiency have attempted to substitute various types of grafts^{1, 2} for valve substance or to occlude the incompetent opening by various types of plastic baffles^{3, 4}. This latter technique has the obvious disadvantage of introducing a foreign body in the heart. We have had initial success with the use of pericardial grafts only to have the graft become fibrotic and ineffective at a later date. This experience with the use of a pericardial graft has been noted by others. Davila and associates⁵ recently reported experimental observations relative to reducing the size of the mitral ring by a circumferential suture about the ring.

The regurgitant flow of blood can be entirely stopped, as determined by the intracardiac finger, by pushing the posterior wall of the annule at the site of the mitral valve forward or anteriorly, thereby decreasing the size of the

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initial ring in an anteroposterior direction. With this concept in mind, anatomic dissections and laboratory experiments were performed in an attempt to devise a technique which would reduce the size of the mitral ring at the posteromedial commissure which, in turn, would approximate the edges of the valve leaflets and obliterate this incompetent space.

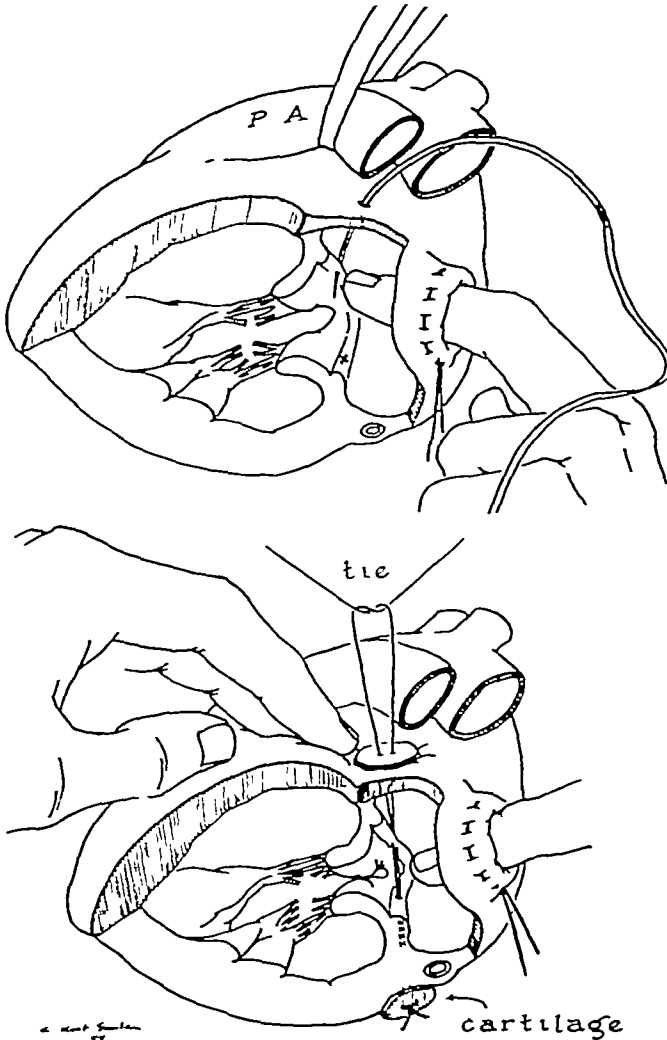


Fig 1—Diagrammatic sketches of the technique of introducing sutures to reduce the size of the enlarged mitral annulus for the correction of mitral regurgitation.

Anatomic dissection of normal hearts and of patients dying of mitral insufficiency showed the incompetence could be relieved by introducing a double suture at the junction of the aorta and auricles through the anterior and posterior segments of the mitral ring adjacent to the posteromedial commissure and out the ventricular side of the coronary sinus in the region free of the circumflex coronary artery posteriorly (Fig 1)

with 73 per cent segmenters, 5 band forms, and 22 per cent lymphocytes. Urinalysis yielded a negative sugar and protein, 4 plus acetone, and an occasional leukocyte per microscopic high power field.

Roentgenograms of the chest exhibited a complete collapse of the right lung with a fluid level at the base. There was no evidence of rib fracture (Fig 1).

Under 1 per cent procaine infiltration anesthesia, an intercostal catheter was inserted into the right third intercostal space in the mammary line and attached to a water seal drainage bottle. A large amount of air was evacuated from the pleural space by this method. Aqueous penicillin, 100,000 units, and streptomycin, 0.5 Gm in 10 c.c. of saline, were placed in the right pleural space.



Fig 1—Roentgenograms show complete collapse of the right lung with a fluid level at the base and complete re-expansion with intercostal catheter in place.

The lung had completely re-expanded in two days and the intercostal catheter was removed. The child has remained well since leaving the hospital.

CASE 2—R L, a 42 year old white man, was admitted to the Good Samaritan Hospital June 26, 1951, because he had been in an automobile accident just prior to admission. He had apparently been in good health before the accident.

Physical examination revealed a well developed, well nourished, acutely ill man who was moderately cyanotic and was having some respiratory difficulty. There were obvious fractures of the right clavicle, right tibia, and left femur.

Examination of the chest showed the left side did not expand as well as the right. The left chest was hyperresonant to percussion. Breath sounds were decreased on the left side. The trachea and mediastinum were shifted to the right.

Pulse rate was 85 with sinus rhythm. Systolic blood pressure was 120 mm Hg, and diastolic pressure 70 mm. Hg. The heart was not enlarged and no murmurs were audible. The remainder of the physical examination revealed no abnormalities.

Laboratory examinations revealed a red blood count of 4,400,000 per cubic millimeter, hemoglobin 12.2 Gm. per 100 c.c. and leukocyte count 12,500 per cubic millimeter with 51 per cent segmenters and 19 per cent lymphocytes. Urinalysis yielded a specific gravity of 1.023, no sugar, 1 plus protein, 2 to 3 leukocytes per microscopic high power field, and 1 to 3 granular casts per microscopic high power field.



Fig 2—Roentgenograms show evidence of a pneumothorax involving the left thoracic cavity with no evidence of rib fracture and complete re-expansion after adequate therapy.

Roentgenograms of the chest exhibited evidence of a pneumothorax involving the left thoracic cavity with the lung being about 80 per cent collapsed. The heart and mediastinum were displaced markedly toward the right and the diaphragm was displaced downward slightly. No evidence of rib fracture was present (Fig 2).

Under 1 per cent procaine infiltration anesthesia, an intercostal catheter was inserted into the left fourth intercostal space in the anterior axillary line and attached to a water seal drainage bottle. A large amount of air was evacuated from the pleural space by this method. Aqueous penicillin 100,000 units and streptomycin 0.5 Gm. in 15 c.c. of saline, were placed in the left pleural space.

The lung had completely re-expanded in four days and the intercostal catheter was removed. The lung has remained expanded and there have been no subsequent pulmonary symptoms.

DISCUSSION

The etiology of traumatic pneumothorax without rib fracture has not been proved to our knowledge. Lundsberg and Liebowitz have stated that nonpenetrating

trauma may produce pneumothorax by bursting injuries to the lung, particularly when the force is applied to an expanded lung with the glottis closed. This would explain Dieffenbach's observation that most of the injuries of this type occur in children in whom the chest wall is more pliable.

The injuries in Cases 1 and 2 can well be explained on this hypothesis.

We have an additional case which we feel had the same mechanism of injury to lung. This case is not included with Cases 1 and 2 as there was evidence of incomplete fractures of several ribs. The fractures were of the green stick type with only the inferior cortex being involved and could not adequately account for the severe injury to the lung. This case also demonstrates one of the more severe types of injury that can result from such a mechanism.



Fig 3—Roentgenograms show incomplete fracture of ribs and pneumothorax on right with re-expansion of lung after adequate therapy.

CASE 3—K T, a 14 year old white boy, was admitted to the Good Samaritan Hospital on June 13, 1952, because he had fallen off a truck and it had run over his thorax. He was unconscious for 10 to 15 minutes. Except for occasional mild infections of the respiratory tract and the usual childhood diseases he had been in good health prior to the accident.

Physical examination on admission revealed a well developed, well nourished, acutely ill boy. There was a laceration of the right ear through the anterior margin across the canal and the entire ear was displaced posteriorly.

Examination of the chest showed the right side did not expand as well as the left. There were abrasions across the right anterior and posterior chest wall. The patient was

coughing up bloody sputum. The breath sounds were decreased over the right lung field. Systolic blood pressure was 100 mm Hg, and diastolic pressure 70 mm Hg. Pulse rate was 110 with sinus rhythm. The heart was not enlarged and no murmurs were audible. The remainder of the physical examination revealed no abnormalities.

Laboratory examinations revealed a red blood cell count of 5 million per cubic millimeter, hemoglobin 13.4 Gm per 100 c.c., and leukocyte count 17,350 per cubic millimeter with 80 per cent segmenters, 14 per cent band forms, and 6 per cent lymphocytes. Urinalysis yielded a specific gravity of 1.025, no sugar or protein, and an occasional leukocyte on microscopic examination.

Roentgenograms of the chest exhibited incomplete fracture of the right fifth and sixth ribs with questionable fractures of the right second and third ribs. The superior cortex of these ribs appeared intact. There was an approximately 60 per cent collapse of the right lung as a result of pneumothorax (Fig. 3).

Under 1 per cent procaine infiltration anesthesia, a 22 French Foley catheter was inserted through the second intercostal space in the mid-clavicular line and attached to a water seal drainage bottle.

The patient continued to pour out large quantities of air through the intercostal catheter. Twenty-four hours following insertion of the catheter he was found to have forced expirations and a respiratory rate of 50 per minute. The trachea was shifted to the left, and the left lung was wet in spite of the patient's effort to cough. His cough was productive of a small amount of bloody sputum.

On June 14, 1952, a right thoracotomy was performed through the right fifth intercostal space anteriorly. There was a moderate amount of blood in the pleural cavity. A large rent was found in the superior surface of the upper lobe which was approximately 12.5 cm. in length and 5 cm. in depth. The posterior segment of the lung was fairly firm and appeared to have a hematoma within the parenchyma of the lung. No active bleeding could be demonstrated. The wound of the lung was closed with interrupted silk sutures. The lung could then be expanded well except for the hemorrhagic portion of the upper lobe and there was no demonstrable air leak. No evidence of bleeding could be found. The pleural cavity was drained with two intercostal catheters, one anterior and one lateral. The pleural cavity was irrigated well with saline, and the wound was closed in layers with interrupted fine silk sutures.

The patient had an uneventful postoperative course and was discharged on the seventh hospital day. The lung remained expanded. The hematoma of the upper lobe gradually cleared over a period of about ten days, and since that time chest films have showed no abnormality of the lung.

SUMMARY

- 1 Three cases of traumatic pneumothorax are presented, two cases without evidence of rib fracture and one with incomplete fracture of ribs.
- 2 Three cases of traumatic pneumothorax have been presented which would substantiate the mechanism of injury suggested by Lindskog and Liebow.

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PERIPHERAL BRONCHIAL ADENOMA OF MUCOUS GLAND TYPE

CLINICAL AND PATHOLOGIC ASPECTS

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SINCE 1882, when H. Muller first described the tumor which has come to be known as bronchial adenoma, two major and opposed theories have been offered to explain its mode of origin. Churchill,¹ in 1937, noted a superficial resemblance of bronchial adenoma to fetal lung and suggested that bronchial adenomas may be vestigial lobes occurring in the bronchial wall. This concept was fully developed by Womack and Graham,² with the intriguing theory that most of the bronchial adenomas were mixed tumors and had an organoid multidermal origin from undeveloped anlagen of the lung. With the passage of time, however, the mixed-tumor theory has lost many advocates and the arguments against it have been convincingly summarized by Liebow³ in these words: "The common appearance (of the tumors) is not that of alveolated tissue, cartilage appears to be engulfed or invaded rather than formed by the tumor and such bone as is found occurs either from metaplasia of pre-existing cartilage or within the septa or capsule of the tumor. It does not seem to be a part of the neoplastic process."

The second major theory, suggesting that bronchial adenoma arises from the bronchial mucous glands or their ducts, was proposed by Reisner⁴ in 1928 and Kramer⁵ in 1930. Both were of the opinion that the tumor cells more closely resembled the secretory ducts of the bronchial mucous glands than the alveoli and were probably derived from the former. Numerous investigators⁶⁻¹³ have concluded that bronchial adenomas of the carcinoid and cylindroid types are derived from the bronchial mucous glands or their ducts. The great bulk of evidence offered in support of this theory has been *indirect* and is summarized as follows:

1 The anatomical location of the tumor in the wall of the bronchus and the presence of intact bronchial epithelium covering it strongly suggested origin from a structure normally found in the bronchial wall. The epithelial nature of bronchial adenoma would logically point to the bronchial mucous glands as the site of origin.

2 Bronchial adenomas apparently never arose from small bronchioles in which mucous glands were absent.

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3 Some investigators have seen "transitional cells" which were thought to bridge the gap between bronchial adenoma and the adjoining bronchial mucous glands. The neoplastic glands, however, did not closely resemble the bronchial mucous glands.

4 The presence of mucus in some of the tumors, either within the lumina of the neoplastic glands or within the cytoplasm of tumor cells, has lent some support for origin in the bronchial mucous glands. It should be noted, however, that the neoplastic glands containing mucus did not closely resemble the normal bronchial mucous glands.

Recently, the first bit of *direct* evidence in support of the theory of origin from bronchial mucous glands has been offered by Ramsey and Reimann¹⁴ who reported a bronchial adenoma which was clearly of mucous gland origin. In that tumor there was a striking resemblance of the neoplastic glands to normal bronchial mucous glands. We have recently observed a similar tumor. These two cases of bronchial adenomas clearly arising in and closely resembling bronchial mucous glands seem to provide a missing pathogenetic link in the theory of origin of bronchial adenomas from bronchial mucous glands.

CASE REPORT

J. H., a 66 year old Negro farmer (retired) entered the District of Columbia General Hospital for the first time on Nov. 9, 1953, complaining of hematuria of eleven days duration. History and physical examination on admission revealed signs and symptoms of prostaticism of several years' duration. The patient underwent a retropubic prostatectomy on Nov. 17, 1953. The pathologic diagnosis was benign nodular hyperplasia.

Chest x-rays taken on Nov. 9, 1953, disclosed an unsuspected solitary nodular density in the right lung at the level of the third right anterior interspace (Fig. 1, A and B). This was interpreted as being due to either neoplasm or granuloma. On Jan. 18, 1954, the patient was readmitted to the District of Columbia General Hospital for resection of the solitary coin-shaped lesion (nonclassified) of the right lung.

Additional history disclosed that the patient smoked one pack of cigarettes a day but denied any symptoms referable to the respiratory system. Tuberculin and histoplasmin skin tests were positive (1 plus).

On Jan. 21, 1954, the patient was operated on and the operative note follows:

Operative Note—The skin incision was made on the right side along the fifth interspace. The pleural cavity was entered through the fifth interspace without removal of a rib. The rib spreaders afforded adequate exposure. Examination revealed a 2 cm. hard, firm nodule deeply embedded in the posterior part of the middle lobe. The tumor could not be seen but could only be palpated. There was no evidence of atelectasis. The posterior part of the middle lobe was freed from the upper and lower lobes to permit accurate approximation of two clamps to permit a wedge resection. The tumor mass was removed with a wide margin of safety. The severed edges of the lung were then sutured with two tiers of 000 chromic catgut on an Atraumatic needle. The first tier was a continuous mattress suture and the second tier was a whipstitch. No bleeding occurred and there was no leakage of air. Upon section of the tumor in the operating room it was believed to represent a metastatic tumor of undetermined origin. No further resection was deemed advisable. Positive pressure anesthesia was applied. The pleural cavity was drained by the water seal method. The chest wall and parietes were closed in layers under positive pressure anesthesia.

Because granuloma was suspected, a portion of the tumor removed surgically was sent to the bacteriologist for cultural studies. These were negative. The remainder of the specimen was submitted to the pathologist for gross and microscopic examination.

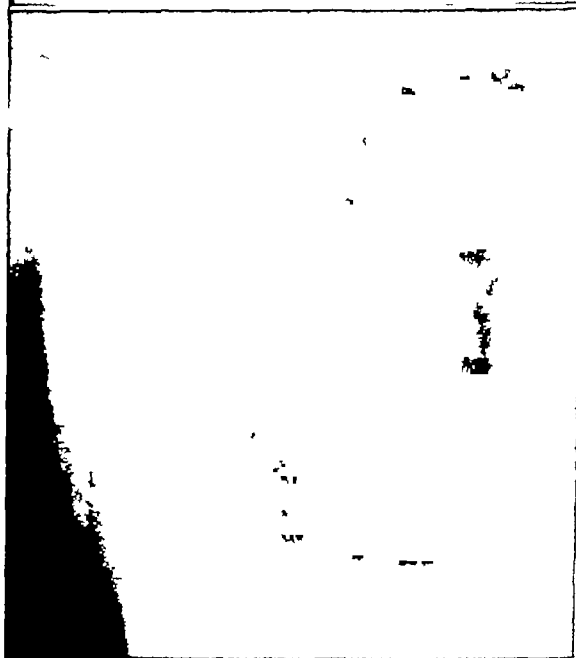
A*B*

Fig 1—Circumscribed solitary mass in the right mid lung field demonstrated by postero-anterior view (*A*) and tomogram (*B*)

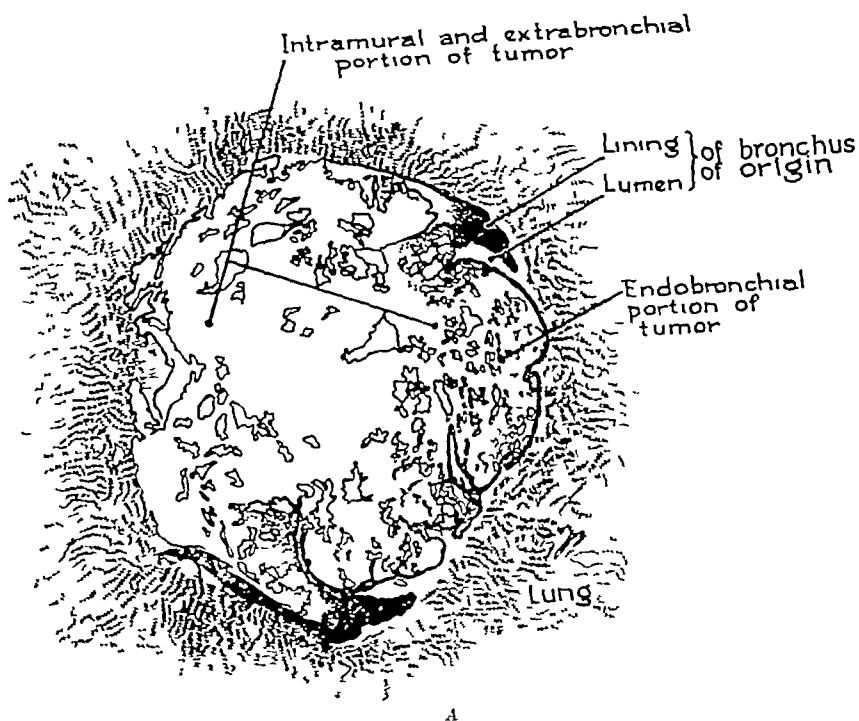


Fig 2—A Diagrammatic sketch of bronchial adenoma of mucous gland type. B Low-power photomicrograph of tumor. Arrows indicate remnants of the lining of the bronchus of origin. Cytically dilated neoplastic glands are interspersed with smaller ones (X35 reduced 1/2.)

Fig 3



Fig 4

Fig 3—Enlarged photomicrograph of section A in Fig 2 B. Below are hyperplastic bronchial mucous glands covered by bronchial epithelium. Above is a portion of the endo bronchial part of the tumor also covered with bronchial epithelium. Note the close resemblance of the hyperplastic mucous glands to the neoplastic mucous type glands above ($\times 150$ reduced $\frac{1}{2}$).

Fig 4—Higher-power photomicrograph from a more central portion of the tumor. Note the larger cystically dilated mucous type glands and the smaller adjoining glands. The nuclei are basal flattened and pyknotic, and the cytoplasm is foamy and rich in a mucicarmine-positive material. Mononuclear cells are present in the connective tissue stroma which surrounds the glands ($\times 160$ reduced $\frac{1}{2}$).

Gross Pathology—The specimen consisted of a wedge shaped portion of right middle lobe taken from the periphery. It measured 4 by 3 by 1 cm. Five-tenths centimeter beneath the pleural surface there was a tan slightly trabeculated, spherical tumor which measured 2.0 cm. in diameter. The cut surface was slightly moist and mucoid. The tumor appeared to be well demarcated from the surrounding lung parenchyma although no true capsule was seen. There was no apparent relationship of the tumor to adjoining bronchi. A frozen section was done because clinically there was a suspicion of metastatic carcinoma, possibly from the previously resected prostate gland. The frozen section diagnosis was "metastatic carcinoma, primary site undetermined." The paraffin section diagnosis was "bronchial adenoma of mucous gland type."

Microscopic Examination—Approximately one third of the tumor lay within a dilated bronchus, while the remaining two thirds was intramural and extrabronchial (Fig 2, A and B). The endobronchial portion was polypoid and covered by pseudostratified ciliated columnar epithelium which was mostly intact. Beneath the lining epithelium of the bronchus and lying in its wall were hyperplastic occasionally dilated mucous glands which were morphologically indistinguishable from the mucous glands comprising the tumor (Fig 3). The tumor was composed almost entirely of mucous type glands between which was a fine areolar connective tissue fairly rich in mononuclear cells and containing lesser numbers of plasma cells and lymphocytes (Fig 4). The mucous glands varied in shape from round and oval to tortuous. They all possessed lumina which contained varying amounts of a stringy mucoid material staining pink with mucicarmine. The lining of the glands was usually one cell in thickness tall columnar epithelium, and the cells had basal nuclei and abundant foamy cytoplasm which stained intensely red with mucicarmine. The nuclei which rested on the basement membrane were small, pyknotic, flattened or ovoid, and closely resembled the nuclei of the bronchial mucous glands. Mitoses were not observed. No ciliated cells were seen. Thin walled blood vessels were present but not in abundance. Within the intramural portion of the tumor was a fragment of degenerating hyaline cartilage, probably a remnant of the cartilage of the bronchus of origin. The tumor was separated from the surrounding lung parenchyma by an ill-defined capsule composed of condensed connective tissue fibers. The small bronchi and bronchioles adjoining the tumor appeared somewhat dilated. A portion of lung parenchyma adjoining the tumor contained many alveoli which were filled with stringy mucoid material resembling the mucoid material seen within the lumina of the neoplastic mucous glands.

CLINICOPATHOLOGIC ASPECTS

We were able to find only one other case in the literature of bronchial adenoma which was pathologically of the mucous gland type and identical with

TABLE I. CLINICAL AND PATHOLOGIC ASPECTS OF THE PRESENT CASE COMPARED WITH THE PREVIOUS REPORTED CASE OF RAMSEY AND REIMAN

CASE OF RAMSEY AND REIMAN	PRESENT CASE
<i>Clinical Comparison</i>	
X ray atelectasis	X ray coin lesion
Bronchoscopy visualized	No bronchoscopy
Signs and symptoms of infection (fever, general aching, etc.)	Asymptomatic
54 years old	66 years old
Race White	Race Negro
<i>Pathologic Comparison</i>	
Tumor arose in main bronchus, right lower lobe	Tumor arose in small peripheral bronchus, right middle lobe
Tumor was entirely endobronchial	One-third of tumor was endobronchial two thirds intramural and extrabronchial
Tumor was 13 cm. in diameter and pedunculated	Tumor was 2 cm. in diameter and non pedunculated
Bronchial adenoma mucous gland type	Bronchial adenoma, mucous gland type
Neoplastic glands were mucicarmine-stain positive (presumptive evidence of mucus)	Neoplastic glands were mucicarmine stain positive (presumptive evidence of mucus)

our case This is the case reported by Ramsey and Reimann in 1953 A comparison of the clinical and pathologic aspects of the two cases is summarized in Table I

The clinical differences may be ascribed to the difference in location of the two tumors The case of Ramsey and Reimann had clinical features which have been described in cases of adenomas located in large bronchi, while the clinical aspects of our case closely resembled those described in cases of bronchial adenomas arising in small bronchi The clinical and pathologic description of bronchial adenomas arising in small bronchi is a relatively recent event and as late as 1948 Willis¹⁵ stated, "Bronchial adenoma is almost invariably situated in a large bronchus close to the hilum, never in the periphery of the lung"

Maier and Fischer¹⁶ have reported five cases of bronchial adenoma arising in small bronchi and three were clinically asymptomatic Good and Harrington¹ reported 23 additional cases arising in small bronchi, 17 of which were asymptomatic All were of the carcinoid type These authors were of the opinion that bronchial adenomas arising in small bronchi close to the hilum of the lung were more likely to produce symptoms than adenomas located at the periphery

The roentgenographic appearance of the tumor in our case (peripheral bronchial adenoma of mucous gland type) closely resembled the roentgenographic appearance of bronchial adenomas arising in small bronchi as reported by Maier and Fischer and by Good and Harrington The x-ray appearance was that of a "solitary intrapulmonary tumor" This designation is used to represent any lesion whether neoplastic, inflammatory, granulomatous, developmental, or vascular that is located within the lung and is round, oval, or slightly lobulated, with a smooth contour sharply demarcated from the normal lung Our studies on approximately 200 cases have indicated the futility of attempting to establish a precise preoperative diagnosis of a circumscribed noncalcified intrapulmonary lesion For this reason, the early use of exploratory thoracotomy as a diagnostic measure is used in all cases of noncalcified circumscribed masses In a series of 156 cases of solitary mass lesions of the lung reported by Good, Hood, and McDonald,¹⁸ 12 (8 per cent) were bronchial adenomas

CLASSIFICATION

The currently accepted classification of bronchial adenomas into carcinoid and cylindroid groups does not include bronchial adenomas of mucous gland type The carcinoid tumors were so named because of their resemblance to carcinoid tumors of the gastrointestinal tract, while the cylindriomas derived their name from a group of benign salivary gland tumors which they closely resembled This classification has been modified by Liebow to include an oncocytoid variant of carcinoid tumors and a mucocystic variant of cylindriomas More recently, the literature on bronchial adenoma includes other "exceptions" to the current classification For example, reference is made to a small round cell variant of cylindroma¹⁹ which resembles "oat cell carcinoma" Two other variants of cylindroma-basal cell carcinoma and epithelioma adenoides cysticum have been described by Holley²⁰

It seems that all bronchial adenomas are channeled into either the carcinoid or cylindromatous groups regardless of how well they fit. It is questionable whether our knowledge of bronchial adenoma is abetted by lumping several pathologically heterogeneous tumors into the carcinoid and cylindromatous groups. We believe that an improved pathologic classification of bronchial adenoma based upon a study of a large number of cases would be valuable.

MALIGNANCY OF BRONCHIAL ADENOMAS

The tumor in our case was unusually well differentiated into mucous type glands and appeared to be entirely benign. No regional lymph nodes were available for histologic study and no nodules were felt in the lungs at the time of operation. Ramsey and Reimann believed that their tumor was also entirely benign. Opinions regarding the degree of malignancy of bronchial adenomas of the carcinoid and cylindromatous types run to extremes. For example, Womack and Graham regarded many of them as potentially malignant and believed that some of the bronchogenic carcinomas arose in pre-existing adenomas by malignant transformation. Wessler and Rabin²¹ and Rabin and Neuhoef,²² on the other hand, regarded most bronchial adenomas as benign lesions.

According to Moersch and McDonald, carcinoid tumors comprise 90 per cent of bronchial adenomas and cylindromas comprise 10 per cent. A difference in the relative malignancy of these two groups has been postulated by McDonald²³ on the basis of a greater infiltrative tendency on the part of the cylindromas, and McBurney, Kirklin, and Woolner²⁴ found that cylindromas metastasized three times as often as carcinoid tumors. Liebow, however, has pointed out that only a very few instances of *distant metastases* of the cylindroid variety have been reported, and he indicated that more data are needed before the question of the relative malignancy of the carcinoid and cylindroid types can be decided.

Recently McBurney, Kirklin, and Woolner reviewed all reported cases of "metastasizing bronchial adenoma." They were able to find 87 cases, of which 46 showed regional node metastasis only. The remaining cases had metastases in order of frequency to the liver, opposite lung, ipsilateral lung, pleura, esophagus and other mediastinal structures, bone and miscellaneous organs including brain, kidney, and adrenal gland. The incidence of metastasis of all reported cases was 10 per cent. The authors did not include data with regard to duration of life following discovery of the tumors and post-mortem findings with regard to the extent of tumor dissemination and the cause of death. We believe that these data are important in evaluating the degree of malignancy of bronchial adenomas. The authors contributed nine cases to the study and none of these included pathologic material or data from post-mortem examination.

The definition of malignancy in cases of bronchial adenoma has been a source of great confusion. Many pathologists and clinicians have considered metastasis to the regional lymph nodes as positive evidence of malignancy even though most cases of this type have had as benign a clinical course as cases without regional lymph node metastases. We believe that "malignant bronchial adenoma" should be defined as an adenoma which will metastasize distantly, and eventually cause death by metastasis.

The recognition of bronchial adenomas which metastasize distantly and cause death by metastasis is beset with many difficulties. Many of the published reports of "metastasizing bronchial adenoma" are deficient in one or more of the following data: (1) post-mortem findings to establish the metastasizing bronchial adenoma as the cause of death, and to rule out (a) a bronchogenic carcinoma arising elsewhere in the lung and (b) an extra-pulmonary malignancy undetected during life, (2) photomicrographs histologically comparing the original biopsy with distant metastases, (3) review of the pathologic material by one or more independent pathologists to establish concurrence in the interpretation of the biopsy or autopsy material, (4) duration of life following the discovery of the tumor.

In conclusion, we believe that many of the cases published as examples of malignant bronchial adenoma are incompletely documented and, therefore, the exact incidence of malignant bronchial adenoma remains undetermined. Carlsen and his associates²⁰ in a recent report of 70 cases of bronchial adenoma have come to similar conclusions.

TREATMENT

No definite conclusions regarding the treatment of bronchial adenomas of mucous gland type are possible on the basis of two cases. Our case was treated by wedge resection because of the peripheral location of the tumor and Ramsey and Reimann's case was treated by segmental resection of the right lower lobe because of the central location of the tumor.

We believe that the treatment of choice in cases of bronchial adenomas of mucous gland type is conservative surgery since the tumors appear to be entirely benign histologically. The type of operative procedure chosen would depend upon the location of the tumor rather than upon the pathologic subtype. Peripheral tumors may be removed by wedge resection, while central tumors may be removed by segmental resection, partial or total lobectomy.

SUMMARY AND CONCLUSIONS

1. A case of bronchial adenoma clearly arising in and closely resembling bronchial mucous glands is reported. The tumor was located at the periphery of the right middle lobe and arose in a small bronchus.

2. The histologic appearance of this tumor lends *direct* support to the theory that bronchial adenomas of the carcinoid and cylindroid types arise in bronchial mucous glands. Most of the evidence previously advanced to support this theory has been indirect.

3. Bronchial adenomas arising in small bronchi are usually asymptomatic and are more readily detected by x-ray than by bronchoscopy.

4. Bronchial adenomas of mucous gland type do not readily fit into the current classification of bronchial adenomas, and other exceptions are noted. There exists a need for reclassification of bronchial adenomas.

5 Bronchial adenomas of mucous gland type appear to be entirely benign. The exact incidence of "malignant" bronchial adenomas of the carcinoid and cylindroid types is undetermined. Many cases so reported are incompletely documented.

6 Treatment of choice in bronchial adenomas of mucous gland type appears to be wedge resection, segmental resection, or total lobectomy, depending upon the location of the tumor with respect to the hilum of the lung.

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THE SURGICAL MANAGEMENT OF FUNNEL CHEST

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SINCE the comprehensive review of the literature on funnel chest by Ochsner and DeBaakey¹ in 1939, the attention of many surgeons has been directed to the problems of etiology and therapy of this deformity. Brown in 1939 first championed the cause of the hypertrophied substernal ligament as an etiological agent, although such a structural defect had been noted by Spitz,² Hofbauer,³ and Hutchinson⁴ many years previously. It was Brown⁵ who first described the minor ligament cutting operation which could be employed successfully in infants, as well as a more extensive cartilage severing operation in the larger defects of older children and adults. The papers of Lester^{6, 8} have very clearly outlined details of the surgical corrective technique which in our hands has proved very satisfactory and effective, and which we have employed with a few minor modifications over the past seven years.

Various other papers⁹⁻¹¹ have reported the experiences of other surgeons with small series of cases employing essentially similar operative techniques with the usual minor modifications. It appears that there is rather universal agreement that the deformity is not as uncommon as once thought, that there is an hereditary factor in the etiology, although some cases occur without familial history of the deformity, that the deformity is usually present from birth, although it does not progress in some instances, and that small deformities can be corrected by a simple operation at an early age. It has been shown¹²⁻¹⁴ that there is definite impairment of pulmonary function in some instances which can be corrected by surgery, and that cardiocirculatory impairment can be produced by extreme degrees of deformity, especially in the older age group. These, too, can be improved by correcting the sternal aberration.

It is obvious, therefore, that further reports of small series of successfully treated cases can be of no great value to students of the funnel chest problem because it has now been adequately demonstrated that there is need for the corrective surgery, and that it can be performed universally with success and with negligible mortality.

It has been our conviction from the time of the early reports that the most confusing aspect of the entire problem has been that of the proper selection of cases and the optimal age for operation. In other words, which cases should we do, and when shall we do them? That problem can be resolved only by observation of large series of cases, of those operated upon and those not operated upon, observed over a period of years.

It has been a universal experience, I am sure, for surgeons all over the country to be confronted by a variety of infants with a variety of degrees of

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thoracic deformity ranging from dimples to deep depressions. One does not ordinarily advise surgery in a very young infant with a very small defect and is rewarded by the disappearance of the dimple in about one-half of these patients and chagrined by the steady growth deepening and widening of the defect in others. The question then is: Does one do 50 per cent unnecessarily or deny surgery unnecessarily in 50 per cent only to regret having procrastinated and thus having lost the opportunity to perform a simple operation instead of the large operation entailing resection of a greater number of costal cartilages? We have not been able easily to foretell at the age of one month which small defect was going to enlarge. In the case of the obviously larger defects there is no problem. As Brown¹⁵ has stated these cannot be done early enough and occasionally the small operation with resection of no cartilage will suffice. Unfortunately we have encountered only two ideal cases for the small operation. In the others the deformity was either too small to warrant surgery or so large that the more extensive operation was mandatory even at a very early age. It has been our experience that infants do not tolerate the extensive resection of costal cartilages necessary in the performance of the large operation, even with adequate fixation of chest wall with anterior struts. It has been seen from our series of cases that deformities classified as moderate to large in size during the first months of life do not ordinarily increase in size appreciably by the first year and delay of surgery until the infant has reached the age of 12 months does not ordinarily necessitate a more extensive operation than would originally have been necessary. The resiliency of the costal cartilages prior to the age of 5 years allows the chest wall to assume rather normal contours once the sternum is elevated. We have not encountered fixed sternal deformities prior to the age of 18 months. If the deformity is markedly symptomatic or increasing rapidly in size surgery is promptly performed.

It has been our observation that the infants under 12 months of age with small defects can be classified roughly into those without upper respiratory obstruction and those with obstruction in the form of congenital hypoplasia of the larynx, congenital stridor, hypertrophy of lymphoid tissue (tonsils, adenoids, etc.) or other causes. It is absolutely mandatory, as Lester¹⁶ stated, that such obstruction be relieved before correction of the funnel chest is attempted. If repair of the tunnel is done in the presence of respiratory obstruction, the likelihood of recurrence of the deformity is great. In a fair proportion of those children with small deformities there will be some diminution in size of the deformity once the obstruction is relieved. In at least two of our nonoperative cases there has been sufficient diminution in size to warrant abandonment of the decision to perform surgery.

On the other hand, our very first case demonstrated that restoration of the normal mechanism of respiration by repair of a funnel deformity was necessary before a 6-week-old infant with laryngeal stridor could breathe sufficiently well to be removed from an oxygen hood. Paradoxical motion of the thorax was marked. There was (Fig 1 A and B) contraction of the lower thorax and both costal arches as well as retraction of the sternum on inspiration prior to operation. Immediately after resection of the seventh and eighth costal car-

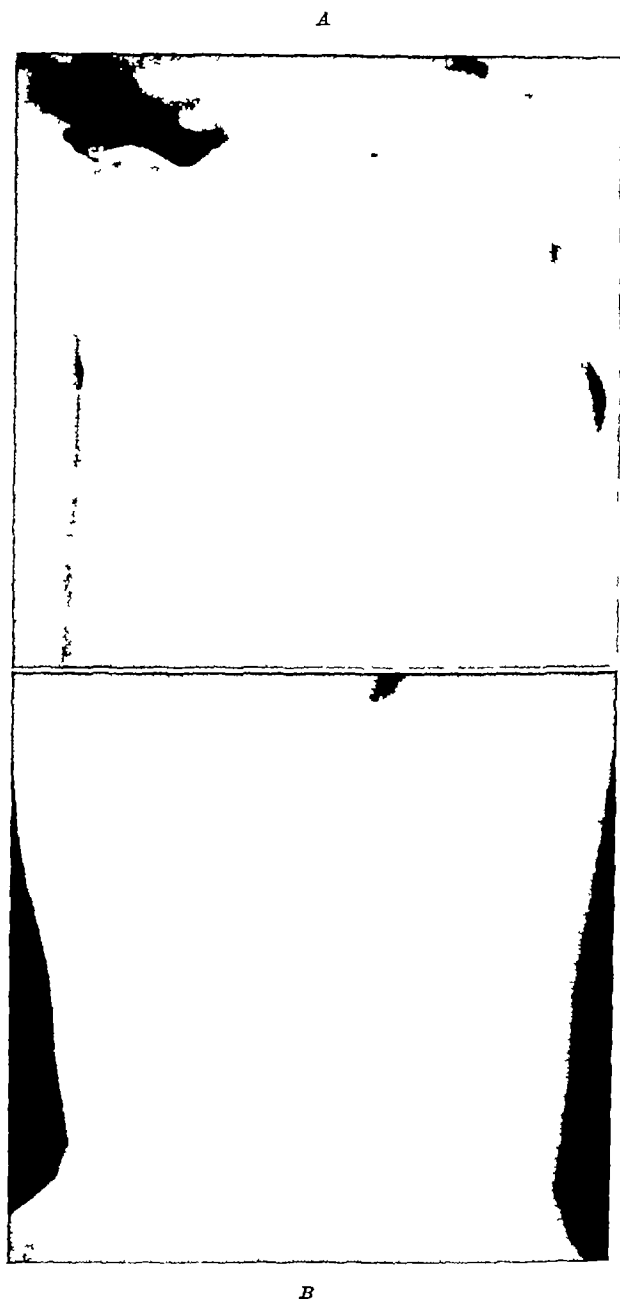


Fig 1—A Photograph of the funnel chest deformity of a six-week-old infant who was unable to maintain oxygenation out of an oxygen hood prior to operation B Photograph of child shown in Fig 1 A two months postoperatively

tilages bilaterally with section of a hypertrophied substernal ligament, the paradoxical motion ceased, and with hyperextension of the neck, the child was able to breathe normally. In this instance, it was felt that the operation was life-saving in that it provided adequate respiratory exchange for the first time since birth. We have had no other cases in which the changes created by operation were so dramatic, although the paradoxical movement of the lower thorax is markedly diminished in all cases immediately following correction of the deformity. In this regard, it may be well to mention that stripping the anterior diaphragmatic insertions downward from the perichondrial beds of the resected cartilages of the costal arch seems a most important part of the operation and is the most effective maneuver in the reduction of diaphragmatic tug on the costal arch and lower thorax thereby greatly lessening paradoxical motion. Even though paradox did not disappear immediately in all instances, it was no longer present by the time the child was discharged from the hospital—a period usually not exceeding two weeks.

The presence, absence, or degree of paradoxical motion of the lower thorax has been one of the influencing factors in reaching a decision whether to recommend surgery or not in the younger infants. In those infants having small defects, with little or no paradox, it has been our policy to withhold surgery and to observe the infant at monthly intervals, carefully measuring the size of the defect. In over 50 per cent of these infants the defect disappeared. It was found that without exception the greater the degree of paradoxical motion, the more likely the defect was to persist or to enlarge. This observation corroborates Sweet's¹⁶ impression on reporting his first two cases. In those cases in which the defect was found to remain stationary, operation was postponed until after the first year of life. In those cases in which there was appreciable increase in the size of the deformity, immediate surgery was done. We have been so impressed by the relationship of paradox to the continued growth of the defect, that we have recently come to urge surgery upon every infant with small defects who demonstrated marked contraction of the costal arches on inspiration. This attitude has resulted from the fact that in no instance in which there was marked paradox did the defect remain stationary. All of such defects continued to enlarge over the period of observation. This factor will undoubtedly result in a larger percentage of early operations in our series in the future. (There have been only five under 6 months to date, as contrasted to ten in the 6- to 12-month age group.)

After the date on which Lester⁶ stated that paradox is present in these patients even when an endotracheal tube is in place, we have routinely taken a moment or two before beginning operation to note that this is universally true, although paradox is not quite as marked in the anesthetized intubated patient as it is when the child is awake and sitting erect.

We have not encountered fixed deformities of the sternum in the age group under 2 years other than the sharp angulation of the xiphoid process. This process has been resected of necessity in almost all instances. After this age, fixed deformity of the sternum of some degree is the rule, and must be dealt with. It is almost needless to repeat the observation regarding posture which

has been made many times before, namely the round shouldered, potbellied profile which accompanies the sternal deformity which becomes noticeable as soon as the child assumes the upright habitus and which becomes more strikingly apparent with each passing month



Fig 2—Photograph of a child having a marked transverse groove deformity accompanying a funnel depression of the sternum

In some instances there are, in addition to the sternal depression, transverse grooves and ridges of the thorax which must be dealt with by the resection of longer sections of costal cartilages and ribs (Fig 2)

The purpose of this paper is to report our observations on 51 operative cases, and ten cases upon whom operation was judged unnecessary at first examination

Risk of Operation—There were no operative deaths. Only one complication, in the form of a minor wound infection, was encountered in the series

The age incidence of surgical cases is tabulated in Table I. It is seen that the largest number of surgical procedures were done in the age group between

6 months and 2 years. As has been mentioned, it is our opinion that the ideal age for surgery is between the twelfth and twenty-fourth months. Probably the bulk of operations done in the future will be done upon this age group. At this age the child appears to be able to better tolerate anesthesia, as well as the paradoxical respiration that occurs following removal of costal cartilages and sternotomy. The fixed deformities of sternum have not yet occurred at this early age, and so waiting until the child reaches this age does not necessitate a more extensive procedure.

TABLE I. AGE INCIDENCE

AGE	NUMBER
Under 6 months	5
6-12 months	10
1-2 years	13
2-3 years	8
3-4 years	5
4-5 years	0
Over 5 years	10
Total	51

TYPE OF OPERATION

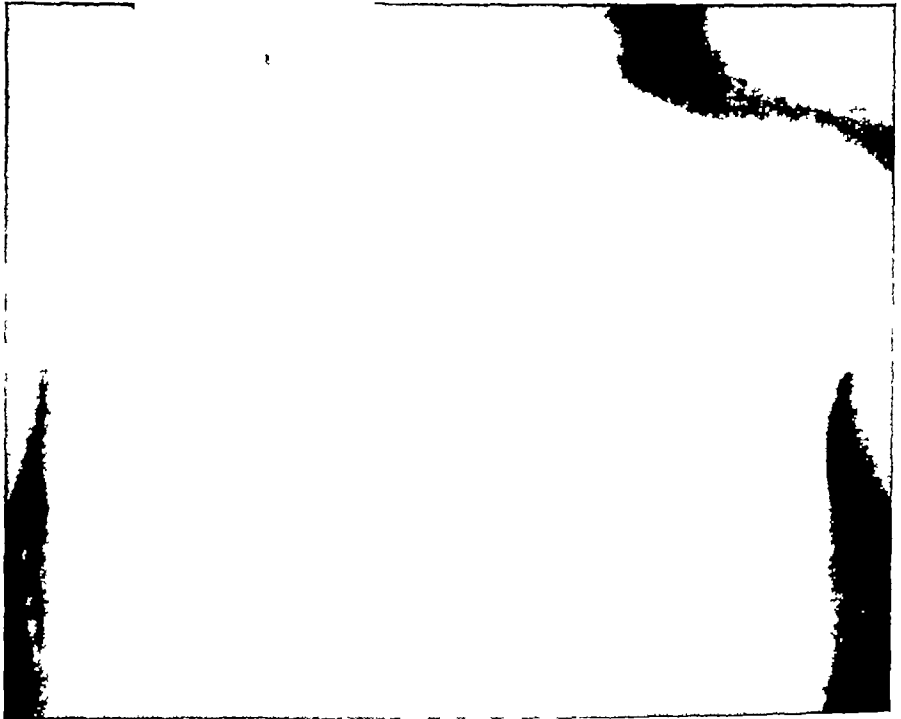
The simple ligament-cutting operation was done in only two instances, both in children not over 4 months of age. One of these patients was described above—our first patient in the series in whom paradoxical motion of the thorax was so marked that prior to operation the child could not be removed from the oxygen tent. Following the simple operation, respiratory movement became immediately more effective, and the child has undergone remarkable growth and development (Fig 1, B).

The only other simple operation was performed upon a 4-month old infant with a small defect. The operation upon this patient was done early in the series and probably at the present time the patient would be observed until the age of 1 year. In this instance a portion of the deformed costal arches was removed bilaterally. The child has done well.

In forty-nine other cases the extensive subperichondrial resection of costal cartilages has been done. All of the deformed cartilage—sometimes this involves resection of the entire cartilaginous portion—of the rib was removed. The number of cartilages removed depended upon the number involved in the deformity and varied from segments of three to six or seven cartilages bilaterally. We have been particularly careful to remove all of the involved portion of each cartilage, preferring to remove an excess rather than an insufficient amount since regeneration promptly takes place if subperichondrial resection is done, and stability of the chest wall returns in a matter of ten days in almost all instances. This procedure, we feel, has been an important factor in eliminating the necessity for second operations for recurrent deformity. There have been no recurrences in the entire series of cases. All cases are being observed at least yearly. The longest period during which any case has been under observation is seven years.

We believe that it is extremely important to perform subperichondrial resection for two reasons: first, regeneration of cartilage or bone occurs rapidly,

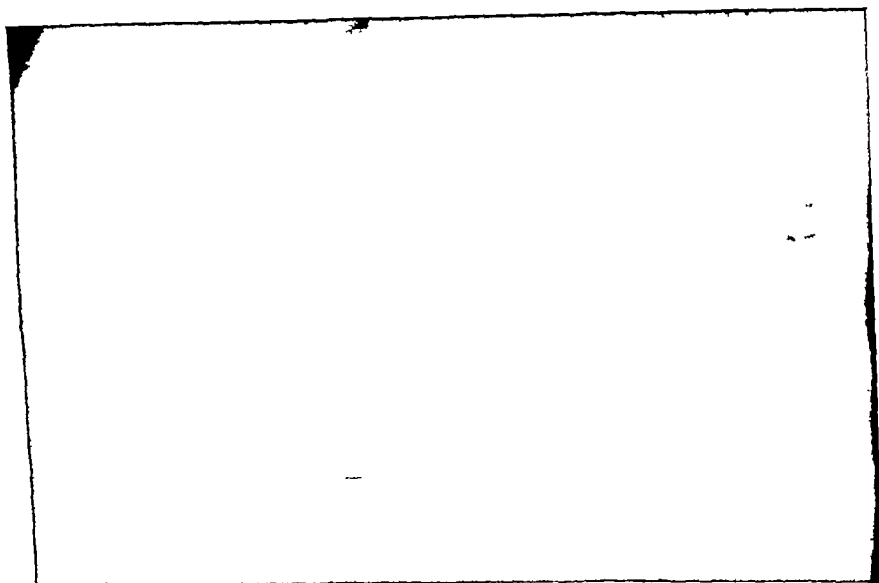
and adequate stability of the chest wall is assured in less than two weeks, second, by judiciously placing sutures in the perichondrium of resected ribs the shape of the chest wall can be altered within limits so that external fixation of the sternum is scarcely necessary to ensure regeneration of cartilage in the corrected position. In other words, the shape given to the anterior chest wall by carefully placed sutures in the perichondrium was made permanent by cartilaginous regeneration.



A

Fig 3—A Photograph of the chest of a four-year-old child two weeks after operation showing adequate correction of the deformity. B Photograph of same child as in Fig 3 A, two years postoperatively showing apparent recurrence of the funnel deformity due solely to poor posture. C Photograph of the same child as Fig 3 A and B two months after physical therapy in the form of posture training, breathing and shoulder exercises showing disappearance of the deformity apparent in Fig 3 B.

Another factor which we have felt to be of great importance in avoidance of recurrence of the funnel deformity is postoperative physical therapy in the form of breathing exercises, posture training, and shoulder exercises. Of course, all of these measures cannot be instituted in the very young age group because of lack of cooperation, but shoulder exercises and posture training can be accomplished even in the 2-year-old. The potbellied, round-shouldered habitus of the child with funnel chest has been described many times, and it has been our experience that the round shoulders and potbelly often remain a long time after the funnel has been corrected. Fig 3, A-C shows a child, 2 weeks postoperatively (A), two years after resection (B), and several months after posture training had been reinstituted (C). In this instance the referring pediatrician had re-

*B**C*Fig 3 *B* and *C* (See legend on opposite page)

turned the patient to us for consideration of a second operation. As can be seen from the photographs simple exercises were the only measures necessary to restore the normal appearance of the chest. There was, in this instance, no deformity of the regenerated cartilages. Physical therapy will not, of course, correct deformed cartilages and is of no value when such deformities exist.

ANESTHESIA

Intratracheal light plane anesthesia with generous amounts of oxygen was employed in all cases as has been our practice in all thoracic surgery at Milwaukee Children's Hospital. We have encountered no laryngeal edema attributable to the tube in any case and have felt extra security in the knowledge that the anesthesiologist was adequately prepared for the inadvertent opening of the thorax which occasionally takes place in these operations. Although positive pressure can be given with a tight-fitting face mask, we have always felt that an intratracheal tube was much more adequate.

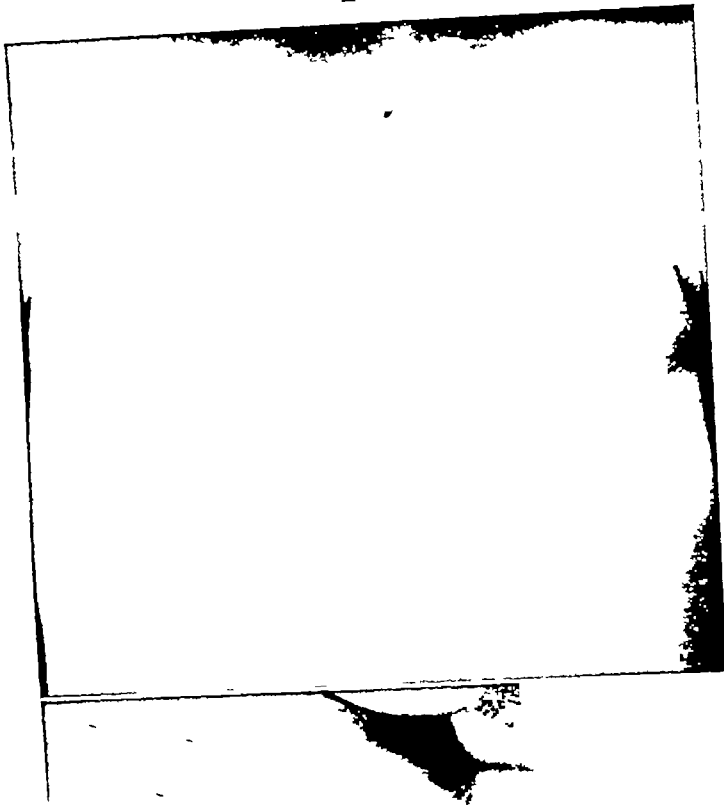
AGE AT OPERATION

It can be seen from Table I that over 50 per cent of our patients were operated upon prior to the age of 2 years. Operations upon the older age groups do not represent postponed surgery in any case, but were done later in life because the patient was seen for the first time at the more advanced age. The oldest patient in the series was 29 years old, operation was done because of impaired respiratory reserve. The result was satisfactory but not spectacular, and there is some question as to the improvement in respiratory function. Maximum breathing capacity increased from 75 to 95 liters per minute (scarcely a spectacular improvement, but the patient is subjectively improved).

SUBSTERNAL LIGAMENT

Hypertrophy of the substernal ligament of significant degree was noted in only thirteen cases in this series. Thickening of sufficient magnitude to cause abnormal traction on the lower sternum was observed in only five cases. A sufficient amount of thickening to warrant the term hypertrophy was noted in eight other cases. In thirty-eight cases there was no appreciable thickening or change in the ligament which differed from the structure observed in autopsy specimens of similar age. It is difficult, therefore, to attribute the deformity to the pull of this ligament on the sternum. It has seemed to us that as Sweet¹⁶ has suggested, "the deformity is primarily skeletal." It appears that there is actually an overgrowth of costal cartilage which in its growth has forced the sternum backward toward the spine in the case of funnel chest, and which in the allied condition of pigeon breast has forced the sternum anteriorly (Fig 4, A and B). Following resection of the deformed cartilaginous segments, the maneuver of elevating the sternum to its normal position reveals that the lateral sternal borders are almost, if not actually, in contact with the ends of the unresected cartilaginous or osseous rib (Fig 5, A and B). This observation would tend to give weight to the concept that the deformity is caused by an overgrowth of cartilage rather than by ligamentous or muscle "pull." There is almost a superfluity of length of periosteum which has covered the deformed resected cartilage. This is usually reefed or "gathered" in the course of the repair, thereby elevating the costal arch and suspending the anterior chest wall from the upper resected ribs in such a way that the wall is quite stable even before regeneration takes place. When regeneration does progress, it proceeds from the perichondrium and periosteum of the resected ribs now lying in the cor

A



B

FIG 4—A and B Photograph of a nine-year-old boy showing pigeon breast type of deformity due to overgrowth of costal cartilages and outward bowing of the cartilages with no deformity of the sternum itself.

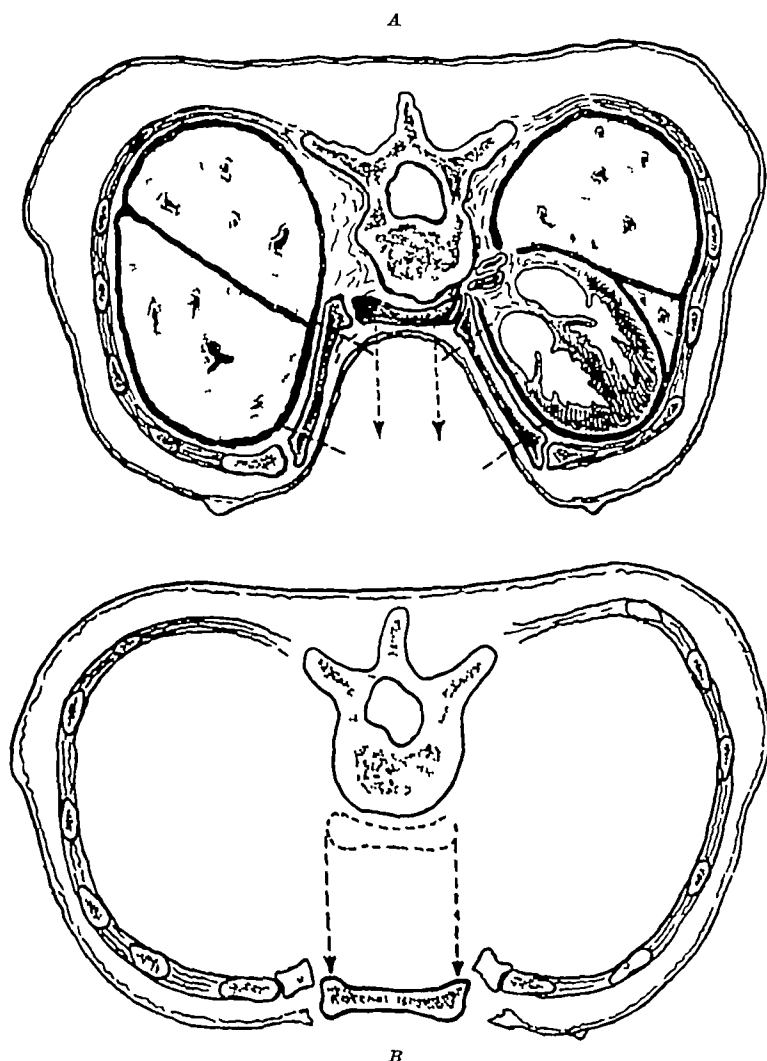


Fig 5—A, Diagram of a cross section of the thorax showing deep depression of the sternum and indicating between the broken line the amount of costal cartilage to be resected. The arrows indicate the distance which the sternum will be moved anteriorly following transverse osteotomy. B Diagram of a cross section of the thorax showing the position of the sternum following resection of deformed costal cartilages bilaterally transverse sternal osteotomy and elevation of the sternum to the corrected position. Note that the lateral sternal margins are almost in direct contact with the short remnants of costal cartilages and in some cases where the entire length of the costal cartilage has been resected are in contact with osseous rib.

rected position. Although no secondary operations were performed which would allow direct inspection of the regenerated cartilages, examination of these cartilages by palpation would indicate that the regenerated segments are somewhat tortuous as a result of the reefing, but that the contour of the chest wall approaches the normal. It is felt that the tortuosity of the regenerated segments adds stability to the chest wall.

AFTERCARE

It is not sufficient merely to correct the costochondral and sternal defects by resection. In those children who have assumed the round-shouldered, pot-bellied posture it is important to institute postural training, breathing and shoulder exercises which are calculated to encourage throwing back of the shoulders, elevation of the thorax and diaphragmatic rather than abdominal breathing. These exercises are begun on the third or fourth day following surgery and are continued for variable periods until such time that the child overcomes his tendency to slouch with consequent dorsal kyphosis and protuberant abdomen.

A course of physical therapy is given in each case until discharge of the child from the hospital, and his exercises are continued at home under the direction of the mother until the child has acquired the habit of maintaining proper posture.

UNOPERATED CASES

During a period of seven years, ten children were seen on whom surgical correction was not deemed advisable (Table II). Two of these had congenital laryngeal stridor. It was felt that the deformity of the chest was secondary to the stridor. In both of these patients seen at the age of 1 month and 17 months, respectively, the deformity subsequently decreased in size to nonsurgical proportions following relief of the stridor.

TABLE II UNOPERATED CASES

AGE AT FIRST CONSULTATION	REASON FOR DELAY OF OPERATION	PRESENT STATUS
1. 1 month	Laryngeal stridor	Minimal deformity
2. 17 months	Laryngeal stridor	
3. 1 month	{ Defect too large for simple operation Too young for large operation	Minimal deformity
4. 1 month	Deformity too small to warrant surgery	Minimal deformity
5. 1 year		Minimal deformity
6. 2 years		Minimal deformity
7. 5 years		Minimal deformity
8. 2 months	Exstrophy of bladder	Deformity still present
9. 9 months	"Celiac" disease"	Deformity still present
10. 2 years	Tetralogy of Fallot	Deformity still present

In the case of two infants seen at the age of 1 month with moderately large defects, on whom the larger operation seemed mandatory, surgery was to be postponed until they had reached the age of 1 year (the age which we considered ideal for the larger operation). Surprisingly, on the follow-up examination at the age of 1 year, the defects had decreased to such an extent that it was felt

surgery had better be postponed until further observation had established whether the improvement were going to continue. Both are now over 2 years old and appear to have only minimal deformities.

Three children, 1 year, 2 years, and 5 years of age, respectively, with very small defects were observed over a period of several years. The deformity did not increase in size in any of these, and although surgery was offered for purely cosmetic reasons, it was refused by the parents.

Three infants who had additional severe congenital defects (tetralogy of Fallot, exstrophy of the bladder, celiac disease) are still being observed, and corrective measures will probably be taken when and if the other problems are resolved.

SUMMARY

Observations gained from the study of fifty-one operated and ten non-operated cases of funnel chest are presented. This group of cases substantiates the fact that morbidity and mortality of surgical correction are negligible. The corrective surgical technique is fairly well standardized and productive of very satisfactory results in most instances.

The criteria for proper selection of cases for operation with particular reference to the ideal age and the type of operation indicated are discussed. The etiological role of the substernal ligament is discussed. In the light of our inability to demonstrate hypertrophy of this structure in most instances, we have been led to conclude that the deformity is primarily skeletal, due to an overgrowth of cartilage rather than to abnormal stresses on the skeleton by hypertrophied ligaments and diaphragmatic muscles. Observations which led us to this conclusion are presented.

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MANAGEMENT OF THE POSTRESECTION SPACE IN TUBERCULOSIS

I FOLLOWING SEGMENTAL AND WEDGE RESECTION

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THE past five years have seen a progressive increase in the popularity of simple excision for residual tuberculous lesions¹ Their characteristic location in the apical, posterior segments of the upper and superior segment of the lower lobes makes resection of these segments a common procedure^{2,3}

Treatment of a restricted pneumothorax, occurring after segmental or wedge resections of the lung is far from standardized Few published reports dealing with this specific problem are available⁴ It would appear, however, that current methods of treatment are not uniformly satisfactory

Since 1951, this institution in cooperation with several others, has been engaged in the systematic resection of residual necrotic foci Late in 1952, our attention was directed to the high relapse rate occurring in patients with annular shadows persisting after long-term chemotherapy had been discontinued Our surgical program then expanded to include not only the randomized closed-negative group, but the so-called open-negative and the usual number of open-positive and salvage cases

As a result of this rather systematic and consecutive experience with resections, we have accumulated in the past three years a moderate number of cases on which the present study is based. This report covers a three-year period, Aug 8, 1951, to Aug 8, 1954, during which 308 thoracotomies were performed on 292 patients This survey includes only segmental and wedge resections Study of residual intrathoracic spaces of those patients who had lobectomies alone, lobectomies plus wedges and segments, or pneumonectomies will be presented in a future report

Following resection of any volume of pulmonary tissue, no matter how contracted or functionless, a space is created which demands filling by adjacent parenchyma In most instances of segmental and wedge resections, when the adjacent lung is relatively normal, space occupation is not a problem The relative expansion of the remaining lung may or may not be associated with physiological deficit Using current methods of estimating pulmonary function, Overholt and his associates^{5,6} found very little over-all change compared to preoperative values This is, in part, due to the fact that essentially functionless tissue is removed Since, in the diseased area, ventilation is reduced almost equally with circulation and oxygen uptake, minimal adjustment is necessary in the postoperative period J J Hirdes,¹⁰ comparing the functional

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sequelae of lobectomy and segmental resections, found there was little difference. Factors, such as the occurrence of postoperative hemorrhage or the formation of a limiting fibrinous exudate over a raw lung surface, may be much more important than the actual amount of tissue removed.

Our definition of a postresection space is a local or restricted pneumothorax characterized by the absence of lung markings on x-ray. Since space occupation may be merely a mechanical problem, depending on the relative size of the residual space and ability of the remaining lung to expand, we have recognized two groups of space problems for the purpose of treatment and prognosis.

It has been stated that a persistent postresection space was tantamount to a bronchial fistula and was, therefore, always a positive space. Aspiration of many of these spaces has shown them to be essentially neutral, or even slightly negative, by manometric pressure determination. Failure of such space to fill during the early postoperative period suggests that either the adjacent lung is restricted by a plastic membrane or intrinsic fibrosis of the lung is preventing expansion. It has also been observed that segments adjacent to those involved with fibrotic tuberculosis may have undergone near maximum distention and early emphysematous changes as a consequence of the contraction of their neighbors. If these segments are salvaged as space fillers, and we make every effort to retain disease free tissue, their capacity for distention may be limited.

In addition to the absence of positive pressure in these spaces, clinical observations do not suggest bronchopleural fistulas. These patients are asymptomatic and a study of serial radiograms reveals that the majority of these spaces shrink and are eventually replaced by organized fibrinous exudate.

The second group of space problems are those with unequivocal evidence of bronchial fistula. As a rule, it is not necessary to utilize pressure determination in the recognition of these pneumothoraces. These patients have a chronic irritative cough, usually productive of streaking with frank old blood or sanguinopurulent sputum. The space, as seen by roentgenogram, rather than decreasing in size, characteristically balloons as much as intrathoracic relations will permit.

Approximately one patient out of five experiences transient space problems. We have had this complication in 61 patients (20 per cent) involving 308 pulmonary resections (Table I).

It is of interest to correlate the disease status of these patients prior to operation with the occurrence of a postoperative space. The majority had minimal to moderately advanced fibrocavitary tuberculosis. About half of the group had no evidence of remaining cavity by serial planigrams, while the remainder were operated on because of a persistent annular shadow. Again, the majority (49 or 83 per cent) had achieved sputum conversion prior to resection as a result of prolonged chemotherapy (6 to 9 months).

As most of this group had susceptible organisms, early or late tuberculous complications were not anticipated. Three patients (0.5 per cent) had positive cultures from a postoperative space. One followed a decortication in which the

TABLE I. INTRATHORACIC SPACE PROBLEMS FOLLOWING SEGMENTAL AND WEDGE RESECTIONS (308 OPERATIONS)
AUG 8, 1951 TO AUG 8, 1954

NO OF PATIENTS	CAVITY STATUS PREOPERATIVELY	SPUTUM BACT PREOPERATIVELY	LOCATION OF SPACE	DATE OF SPACE APPEARANCE POSTOPERATIVELY
61	Closed 29	Neg 49	Apical pneumothorax 41	1st wk 49
	Open 32	Pos 12	Apical Hydropneumothorax 1	2nd wk. 9
			Apical Hemopneumothorax 8	
			Lateral pneumothorax 3	40 d 1
			Basal pneumothorax 3	9½ mo 1
			Total pneumothorax 2	3 yr 1
			Combined (Apical and basal pneumothorax) 3	
			Recurrent combined 1	

patient had had a positive pleural effusion preoperatively. The remaining two were only single colony growths and did not present any clinical problem. Several of the most troublesome complications occurred in the closed-negative or so called target point patients. It is our impression that the formation of a postresection space is largely a mechanical phenomenon and has no direct relation to the state of the patient's disease. We are in complete agreement, however, with the ever-present danger of bronchopleural complications in the open positive patient with resistant organisms.¹¹

Location of the persistent or recurrent pneumothorax was apical or apical posterior in 41 cases (67 per cent), paralleling the resected area (Table I). It may be mentioned that we routinely use two thoracotomy tubes. The tubes are placed on 12 to 18 cm of negative pressure. The 8 cases of hemopneumothorax were of minor extent, consisting of apical hematomas or blood caps.

Resection of the superior segment or wedges from the lower lobe is prone to result in a lateral pneumothorax or basal supradiaphragmatic space. In 2 patients, the lung expanded well, but after seventy-two hours a total pneumothorax was noted. Both had fibrotic emphysematous lungs which were almost incapable of sealing peripheral leaks.

There were three instances of bilateral pneumothorax. Review of those cases in which bilateral resections were performed for all palpable disease suggests that the occurrence of a postresection space on the first side is not invariably accompanied by the same complication on the opposite side.

The typical apical pneumothorax (80 per cent) will be apparent during the first or second postoperative week. Almost daily x-rays facilitate early recognition and treatment of these spaces, particularly when, from the appearance and behavior of the lung at operation, they may be anticipated. Manifestation of a pneumothorax, attributed to the resection, is uncommon after the second postoperative week. Late development of a pneumothorax or hydro-pneumothorax is more likely to be associated with a bronchopleural fistula of tuberculous etiology. In our 3 cases of late fistulas, however, no evidence of tubercle bacilli was found in the drainage material from these spaces.

Fig 1

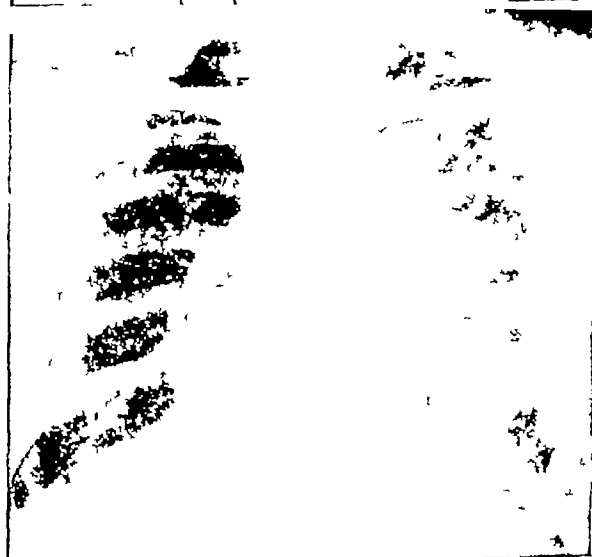
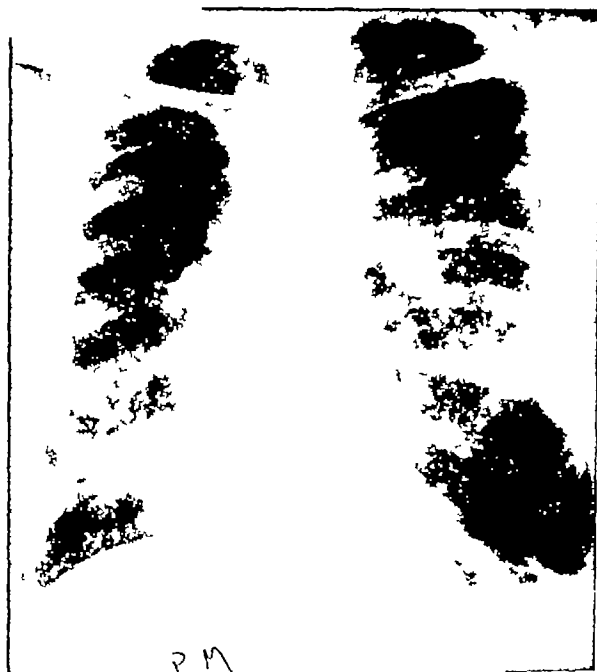


Fig 2

Fig 1—Neutral left apical space following segmental resection
Fig 2—Same space three months later No treatment.

Satisfactory management of the postresection space challenges the judgment of the surgeon and the various methods of treatment in current use

As indicated in the group showing no clinical evidence of a positive pneumothorax or bronchopleural fistula, this suspicion is confirmed by aspiration of the space. Furthermore, delay in space occupation may occasionally be anticipated from operative findings. In this situation, we feel that observation or careful neglect is adequate (Figs 1, 2, and 3)

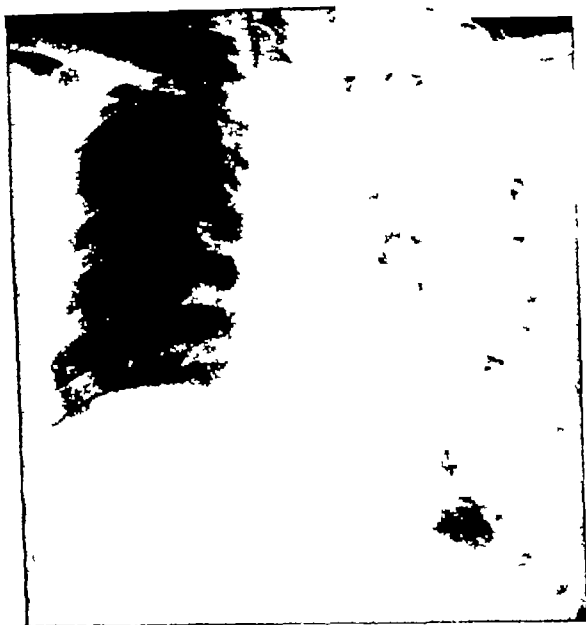


Fig 3—Left space almost obliterated. Small right apical postresection space.

On the other hand, the average space appears following removal of the original thoracotomy tube and represents a local tension pneumothorax, with a communicating fistula of varying size. This type of space, in our experience, is best treated with intercostal catheter, underwater-seal drainage on gentle negative pressure. Two-thirds of our series were successfully managed by single or multiple tube insertions (Table II)

Two adjunctive measures may be mentioned in connection with tube decompression of the local pneumothorax. The Trendelenburg position, if tolerated, is often utilized early in the postoperative period with the hope that the remaining lung may adhere to the chest apex. A second measure, particularly useful for lateral-basal (supradiaphragmatic) or apical-basal spaces, is the induction of a pneumoperitoneum, provided the diaphragm is not fixed (Figs 4 and 5)

We have been most hesitant to use enzymatic débridement in the treatment of a small hemopneumothorax.¹² Although it is claimed these enzymes do not cause fistulas, we are uncertain whether they contribute to their persistence.

FIG 4



FIG 5

Fig 4—Right basal pneumothorax following superior segment resection
Fig 5—Prompt resolution of space with intercostal tube aspiration

Catheter drainage on underwater suction was successful in permitting spontaneous closure of the fistula and reduction of the space by the end of the week in 73 per cent of our group. At the end of this period, the presence of a fistula, virtually undiminished in size, raises several alternatives of treatment.

It is our impression that, after the second postoperative week, the boundaries of the local pneumothorax are relatively fixed. Subsequent organization of the exudate increases the rigidity of the space. At this time, many surgeons will elect to perform open thoracotomy, with attempted closure of the fistula, particularly if the leak is of appreciable magnitude. We have carried out secondary thoracotomy only four times. Three were satisfactory, the fourth recurred but responded to closed drainage. Although the fistula may be surgically closed this does not guarantee obliteration of the space. In addition an involving drainage tube may invite pleural complications, both empyema and subscapular-space infections having resulted from attempted direct fistula closure.

TABLE II. TREATMENT OF SPACE PROBLEMS FOLLOWING SEGMENTAL AND WEDGE RESECTIONS

TYPE OF TREATMENT	DURATION			INFECTION (PREDOMINANT ORGANISM)	STATUS OF TUBERCLE BACILLI (N.T.A.)	
No treatment	6	1 wk.	35	Subtotal (space) empyema	Inactive	42
Thoracentesis	2	2 wk.	10	Proteus vulgaris	1 Active	4
Intercostal catheter	30	3 wk.	4	Friedländer's	1 Negative (In hosp.)	12
Multiple Intercostal tube	10	4 wk.	7	Mixed Proteus and tubercle bacilli	1 Deceased	1
Thoracentesis and tube	2	1½ mo	2			
Secondary thoracotomy	4	2 mo	4	Tubercle bacilli	3 No follow up	2
Thoracotomy and Intercostal catheter	9	4 mo	2	Hemolytic Staphylococcus aureus	11	
Pneumoperitoneum	6	5 mo	3	Total (Staphylococcus) empyema	2	
Streptokinase streptodornase	6					
<i>Result of Space Therapy—</i>						
Satisfactory, 50			Cured 9		Unsatisfactory, 2	

We have preferred to use the postresection thoracoplasty as an aid to space and fistula closure when intercostal tube decompression was unsatisfactory. Failure of intubation is usually apparent during the second to the fourth week.

There is definite morbidity associated with prolonged tube drainage. Intubation beyond two weeks almost invariably results in a low-grade infection of the space. Occasionally, pathogens such as *B. proteus* and *B. pyocyaneus* are present (Table II). These patients complain of intermittent fever and transient discomfort in the shoulder and anterior chest. Also, they are plagued with the well-known systemic handmaidens of a chronic local infection. The mental depression associated with attachment to a tube and bottle drainage for many weeks is difficult to estimate. As a rule, these patients complain of many apparently functional problems. Disregarding the elements of infection, prolonged intubation may accomplish gradual shrinkage of the space and fistula closure. Perhaps some of this is due to the layers of fibrinous exudate

resulting from the infection. Several spaces have reached the unsatisfactory point of diminishing returns when essentially no change is seen from week to week.

We have had the greatest success performing thoracoplasty after a two or three-week trial of intubation when the space is still somewhat collapsible and is located in the apical posterior hemithorax. Thoracoplasty on top of a rigid space, walled with layers of connective tissue, accomplishes little, as with chronic, thick-walled parenchymal cavities.

The majority of space infections were nontuberculous and appeared to be a direct accompaniment of prolonged intubation (Table II). Hemolytic *Staphylococcus aureus* was the common organism, being in all cases penicillin resistant but usually sensitive to erythromycin. Since in most cases the space infections were adequately drained, the only concern was maintaining tubal patency. The infections with other secondary organisms or those in which tubercle bacilli were cultured usually responded to the time-tested principle of space obliteration and were not serious problems. Two total empyemas (*Staphylococcus*) were treated in the standard manner with drainage and subsequent partial thoracoplasty.

Results of space treatment have been classified as satisfactory, cured, or unsatisfactory (Table II). A small apical peripheral rim or cap pneumothorax which progressed to obliteration in several months was accepted as a satisfactory result. Of the unsatisfactory cases, both developed as a consequence of unduly prolonged intubation. Follow-up of this particular group was carried out to relate the occurrence of a space complication to possible late sequelae, and to the status of their tuberculosis (Table II). Four patients who are currently known to have active disease were reviewed, and no apparent relation to the pneumothorax was observed.

DISCUSSION

Proper management of the postresection space is necessary because of the ever present danger of infection and pulmonary hemorrhage. A chronic, undrained intrapleural space is similar in behavior to a parenchymal lung abscess with many of the same clinical symptoms. Even the neutral space, if occupied by lung, has infection potential. Since in most instances the presence of a pneumothorax is due to a bronchial fistula, obliteration of the space requires fistula closure.

The main contributing factor in the pathogenesis and persistence of postoperative bronchoalveolar leaks is the physical state of the remaining lung. Fibrotic lungs, with early emphysematous changes, are more likely to develop a postresection pneumothorax. Tube suction, in such instances, may achieve little toward space obliteration. Postoperative hematomas increase the anticipated incidence of bronchial fistulas, yet a most important factor is loss of the pneumatic function of the lung.

A postresection space, with a small fistula and residual lung capable of moderate expansion, will usually close within the first two postoperative weeks. Beyond this interval, although intubation alone may cause eventual fistula

closure, thoracoplasty or secondary thoracotomy may be a more reliable and earlier expedient. Secondary thoracotomy carries with it the danger of a disseminated intrapleural infection and associated subscapular space involvement. In addition few thoracic surgeons will claim they have had any more than 50 per cent success in secondary bronchial stump closure. As with many surgical problems, there is no standard program, each requiring an individual solution.

SUMMARY

A review of 61 postresection pneumothoraces, occurring in 308 segmental and wedge resections is presented. No relationship was found with the preoperative cavitory or bacteriological status of the patients. The location of the space paralleled the operative site of resected tissue. The majority of pneumothoraces appear within the first two weeks and respond to underwater suction if promptly diagnosed and intubated. A small number of spaces became chronic problems with progenic infection. In the presence of a relatively fixed space early thoracoplasty or secondary thoracotomy may be required, in addition to tube drainage for ultimate definitive therapy. The occurrence of this complication does not appear to influence the future status of the patient's tuberculosis.

Addendum—Since adopting the technique of approximating raw surfaces, we have had no postresection spaces in the past 75 thoracotomies.

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RECURRENT TRACHEO-ESOPHAGEAL FISTULA

(CASE REPORT)

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THE literature of recent years has contained numerous reports of the successful surgical correction of congenital esophageal atresia with tracheo-esophageal fistula. A review of these reports indicates that recurrence of such fistulas is relatively rare, and in several of the larger series this particular complication does not appear. Several authors have suggested that localized suture line leaks might produce stricturing, and following this the reformation of a fistula might be anticipated.

The few recurrent fistulas reported have been recognized in the early postoperative period and it seems that a rather striking clinical change in the patient has led to this recognition, usually in association with symptoms heralding a leaking site. Lyon and Johnson,⁹ in 1948, reported the reopening of a fistula on the ninth postoperative day. Treatment by dilatation was unsuccessful. Autopsy revealed a congenital stricture distal to the site of the fistula which had been overlooked at surgery. Bigger,³ in 1949, reporting on the treatment of 11 patients, mentions briefly one child who died following operation for a recurrent fistula which was recognized seven months after the initial operation. Potts¹⁰ report of 35 cases in 1950 does not mention recurrence of fistula. Recurrence has not been worthy of mention in the extensive experience of Gross,⁴ though he does mention the fact that difficulties in swallowing and recurrent bouts of pneumonitis tend to plague this field of surgery. Abbott and Hopkins,¹ in 1951, reported 2 instances of recurrent fistulas in a series of 20 patients. One of these patients survived the second repair. Kopp and associates,⁷ in a recent report from the University of Pennsylvania, reoperated for leaking anastomotic site in 6 patients of a series of 61, but recurrent fistula was not noted in any instance.

The rarity of recurrence of tracheo-esophageal fistula following primary repair of the congenital defects would seem to render the following case worthy of report.

CASE REPORT

History of Present Illness—The patient was a 30 hour old white boy, born at Walter Reed Army Hospital on May 27, 1952, at 7 38 A.M. Delivery was spontaneous. The mother was gravida iv, para iii. The serology was negative and, aside from polyhydramnios, her pregnancy was normal. The gestation period was 37½ weeks and the child weighed 6 pounds, 13 ounces. He breathed spontaneously. After three minutes in the oxygen hood, cyanosis and apnea were noted and tracheal aspiration and oxygen were necessary to revive the child. Rhonchi persisted throughout the day. The baby's temperature was variable throughout the day, rising to 101° F. At 6 00 A.M. he was offered glucose water.

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He took $\frac{1}{2}$ ounce, promptly regurgitated, and turned deeply cyanotic. He seemed to have a great deal of difficulty in swallowing. An attempt was made to pass a Levin tube, but obstruction was met at approximately $3\frac{1}{2}$ inches from the lips.

Physical Examination on Admission—The patient was a well-developed, well nourished, white, newborn boy with circumoral cyanosis. The hands and feet were slightly cyanotic. Otherwise the physical examination showed essentially normal findings.

Laboratory Data on Admission—The red blood count was 4.8 million with hemoglobin of 15.1 Gm. On May 28, 1952, a fluoroscopic examination of the esophagus, by means of Lipiodol injection through a catheter in the hypopharynx, revealed an obstruction in the proximal esophagus at D₄. The distal end of the obstruction was conical in shape and apparently represented the end of the blind pouch. An upright film of the chest showed the lung fields to be clear (Fig 1). The tracheobronchial tree was outlined by aspirated Lipiodol.

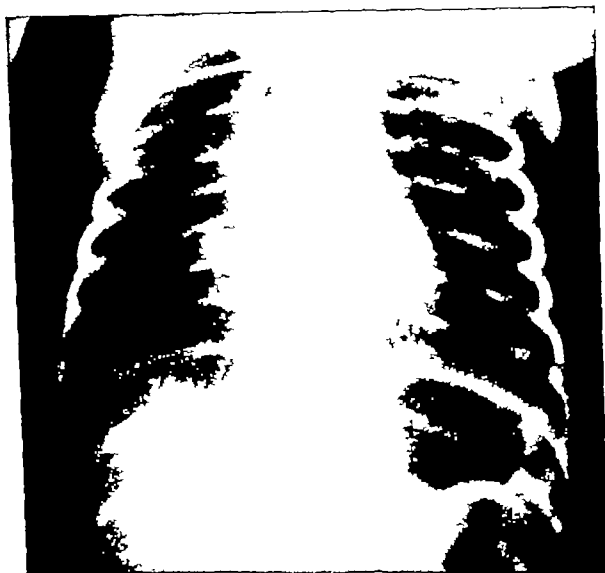


Fig 1—Initial x-ray May 28 1952

The cardiac contour appeared to be within normal limits. The right lobe of the thymus was slightly enlarged and the remainder of the chest appeared to be within normal limits. There was a large gastric air bubble which indicated that the distal esophagus communicated with the trachea. There were no other significant findings. Impression: congenital atresia, proximal one third of esophagus with tracheo esophageal fistula involving the distal segment of the esophagus.

Operation—May 28, 1952—Ether anesthesia was administered by pressure mask. A right thoracotomy was performed through a periscapular incision through the bed of the fourth rib. The extrapleural route was attempted, but the pleura was inadvertently opened in the apical area. After ligation and division of the azygos vein, the distal esophagus was found attached to the trachea just cephalad to the carina. The tracheo esophageal junction was occluded with a Potts' clamp and the esophagus freed from the trachea. The tracheal opening was closed with a continuous suture of No 5/0 arterial silk. The proximal blind pouch of the esophagus was then freed and pulled down into the right hemithorax. An opening was made in the most dependent portion of this pouch and end to end anastomosis between the proximal and distal esophagus was effected with interrupted sutures of No 5/0 arterial silk. One small catheter was placed through the chest wall and attached to a water seal bottle. The patient withstood the procedure well.

On May 31, 1952, a Stamm gastrostomy was performed under ether anesthesia.

X rays—June 2, 1952—A chest film revealed a triangular zone of opacity in the superior aspect of the right hemithorax, corresponding to an atelectatic right upper lobe

Hospital Course—The patient gradually gained weight but suffered repeated bouts of pneumonitis. The recurrent pneumonitis responded to antibiotics. There was no indication of a normal swallowing reflex and attempted oral feeding resulted in aspiration.

Six months after operation, in November, 1952, laryngoscopy revealed an infantile epiglottis without evidence of airway obstruction. False cords and true cords were normal and both sides were mobile. When the patient was in spasm the glottis closed tightly. This examination could not explain the aspiration of food. Impression: no disease of the larynx or hypopharynx. Repeat chest films made during this prolonged period of hospitalization continued to reveal atelectasis of the right upper lobe.



Fig 2—Lipiodol swallow Dec. 11 1952

Lipiodol study of the esophagus performed on Dec 11, 1952, revealed no definite evidence of fistula on fluoroscopic examination (Fig 2). The cardiac shadow and mediastinal contents were retracted to the right. The films at this time showed small flecks of Lipiodol in the bronchus at the level of T₁. It was thought that these flecks were the result of aspiration, as communication between the trachea and esophagus was not visualized.

The patient continued to gain weight and appeared normal except for persistent inability to swallow.

Fluoroscopy was repeated on March 27, 1953. At this time Lipiodol entered the stomach without delay, but a small constant deformity, suggestive of traction diverticulum, was noted in the mid esophagus just above the cricoid.

X rays taken on May 6, 1953, revealed atelectasis of the right upper lobe with the mediastinum drawn to the right. Otherwise the chest appeared within normal limits.

The child's general condition was good and he was discharged from the hospital to continue gastrostomy feedings. The repeated brief attacks of pneumonitis were attributed to the chronic atelectatic state of the upper lobe of the right lung, but the major item of interest throughout this prolonged period of observation was the child's inability to swallow. The interval follow up of the patient reveals additional hospital admissions in 1953 for diarrhea and for dehydration. On each occasion the response to treatment was prompt. In November of 1953 the child was observed for 48 hours in the hospital because of blood flecked vomitus. Outpatient follow up was continued until March, 1954, when definitive study of the esophagus was deemed necessary to clarify the cause of the child's inability to swallow. Throughout the period from May, 1953, to March 31, 1954, the patient seemed to develop normally though slowly.



Fig 3—Demonstrates fistula April 5 1954

The patient was hospitalized again on March 31, 1954. Physical examination at that time revealed a well developed, well nourished, small, alert, active boy weighing 18 pounds, 10 ounces with a temperature of 99° F. The tongue was maintained in a position of partial protrusion and the throat was normal. The chest was symmetrical with a healed periscapular scar on the right side. Auscultation of the chest revealed suppression of breath sounds over the right upper lobe, but no râles were heard. The heart was within normal limits by percussion and auscultation.

Laboratory Data—The red blood count on admission was 3.6 million with a hemoglobin of 9.1 Gm and the white blood count was 23,150 with 73 per cent neutrophils, 21 per cent lymphocytes, 2 per cent monocytes and 3 per cent eosinophils. Urinalysis was negative. On April 14, 1954, the white blood count was 32,600 with 10.5 Gm of hemoglobin. X-ray taken on April 1, 1954, revealed considerable improvement in the appearance of the right upper lung field. Esophagoscopy was performed without anesthesia on April 2, 1954. A 5 mm by 35 cm esophagoscope was passed into the hypopharynx and

through a normal cricopharynx. At a distance of 15 cm from the incisor teeth, the esophagus narrowed to a small opening postero inferiorly and to the left, and to another opening visualized anterolaterally to the right. The opening postero inferiorly was the larger and would admit a No. 16 French bougie which could be passed for approximately 5 cm, at which point resistance was met and further passage prevented. Normal mucous membrane appeared to cover both orifices. Diagnosis: atresia, congenital esophagus with possible residual broncho esophageal fistula. X-ray studies were performed on April 5, 1954, and Lipiodol was introduced into the gastrostomy tube in the stomach (Fig. 3). The patient was placed in the Trendelenburg position. Manual palpation and multiple positioning were done, but no Lipiodol could be refluxed into the esophagus. A catheter was introduced nasally into the esophagus which passed readily down to the cardiac end and a small amount of Lipiodol was inserted. No abnormality of the lower end of the esophagus was noted. When the catheter was withdrawn to the level of the coryna, a small amount of Lipiodol was again injected. Almost immediately a small globule appeared in the right main stem bronchus. Following a short delay the Lipiodol was seen in both main stem bronchi. Impression: tracheo esophageal fistula, probably just proximal to the coryna, with stricture of the esophagus at the operative site. The stricture was approximately 1 cm long.

Operation—April 15, 1954. The anesthesia was accomplished with gas oxygen ether administered via endotracheal tube. The patient's temperature was reduced to 93° F by use of an ice mattress. A right thoracotomy was performed following excision of the existing periscapular scar and the fifth rib was excised. A generalized pleural symphysis was encountered. The right lung was mobilized by sharp dissection. Dissection of the mediastinum revealed a thick walled fistulous communication arising from the posterior wall of the trachea approximately 15 cm proximal to the carina and attached to the anterior wall of the esophagus at the site of the previous end to end anastomosis. The fistula was approximately 1 cm in length. It was transected and the esophageal tracheal ends were closed with interrupted sutures of No. 3/0 black silk. A portion of the anterior mediastinal pleural flap and attached areolar tissue was placed over the tracheal suture line and anchored to the posterior tracheal wall, effecting interposition of tissue between esophageal and tracheal suture lines. The patient withstood the operation well. Rewarming was gradual over a two hour period following the completion of the operation.

Postoperative Course—Postoperative gastrostomy feedings were continued for ten days. Penicillin was given for fourteen days. The patient was started on purées on the tenth postoperative day and was gradually extended to a full diet which he tolerated well and without difficulty. He was gaining weight at the time of discharge on the eighteenth postoperative day at which time films revealed satisfactory clearing of the upper lobes of the right lung. Late follow up films taken on Aug. 2, 1954, showed no evidence of tracheo esophageal communication.

DISCUSSION

The recurrence of fistula following primary surgical treatment of esophageal atresia with tracheo-esophageal fistula is a rarity. Several factors might lead to the re establishment of such a fistula: (1) a leaking esophageal anastomotic line with direct extension into the site of tracheal closure, (2) a direct contact between the adjacent anastomotic suture line of the esophagus and the suture line in the tracheal wall, (3) mechanical trauma resulting from early postoperative dilatation of the esophagus.

The contiguous position of the suture lines and a localized leak of the esophageal suture line would appear to have been the factors in the establishment of the fistula reported here. The patient's true pathology was masked by recurrent pneumonitis and a diagnosis of faulty deglutition for a period of

months until the existence of the fistula was proved. No doubt the repeated courses of antibiotics kept the child alive and also aided in tentatively substantiating the diagnosis of recurrent pneumonitis. The radiological studies performed during the first year of life were at best equivocal, but since a patent esophagus was demonstrated these tended to discount the possibility of a fistula. In view of the repeated prompt response of the upper respiratory symptoms to antibiotics the equivocal x-ray studies and the child's inability to swallow it is understandable that such a clinical course might be interpreted as in the case reported in such a way as to confuse the need for esophageal surgery in infants.

Many accurate descriptions have been written dealing with the diagnosis of tracheo-esophageal fistula with esophageal atresia. The existence of a fistula without esophageal atresia or stenosis is not a familiar picture and consequently may be overlooked. Holt and co-workers⁶ in 1946 and Leigh and associates⁷ in 1951 directed attention to the difficulties which might delay or prevent the diagnosis of tracheo esophageal fistula in the absence of esophageal obstruction. Such instances as have been reported exhibit similarity in the fact that they have masqueraded under the combined diagnosis of faulty deglutition mechanism and recurrent pneumonitis. Haight⁸ has documented the details of a case in which fistula was suspected at birth but the diagnosis was not confirmed until the patient was 4 years old, when surgical correction was carried out. Leigh and associates⁷ in their report included 2 patients in whom the diagnosis of fistula without atresia was confirmed. Both these reports have suggested the use of additional radiological studies not generally used in the routine evaluation of tracheo-esophageal fistula with atresia. Holt⁶ has been impressed with prone-position esophagograms while Leigh⁸ was able to demonstrate fistulas with tracheograms made with the patient in a reclining position. Certainly it seems justified to include these two radiological studies when evaluating a prolonged postoperative course of any infant following anastomotic surgery for tracheo esophageal fistula with esophageal atresia. Such studies might be of great value in the diagnostic survey of any infant suffering repeated bouts of pneumonic infection either with or without evidence suggestive of faulty deglutition.

The reports dealing with operative technique generally have not stressed any particular point designed to prevent the recurrence of fistula. Haight⁸ in 1948, recommended the use of a pedicle flap of mediastinal fat and areolar tissue to buttress the suture lines following the closure of tracheo-esophageal fistula. He called attention to the possibility of the recurrence of a fistula at the sites where the esophageal and tracheal suture lines were in close apposition. Albi and Gilchrist² in the following year, discussing tracheo-esophageal fistulas resulting from blunt trauma suggested that the interposition of a mediastinal flap between the suture lines might prevent the recurrence of a fistula at the site of closure. The use of an interposed mediastinal flap should be a routine part of operative technique in the treatment of tracheo esophageal fistula regardless of the etiology of the fistula.

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AN UNUSUAL CASE OF COARCTATION OF THE AORTA

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THE development in fetal life of the vascular trunk with the great vessels is based on a double anlage with subsequent reduction. The aorta caudal to the brachial arches becomes fused as an unpaired dorsal aorta, and the left fourth arch persists as the definitive aorta. For this reason congenital anomalies have a fairly high incidence. Eek,¹ Gross and Ware,² later Apley³ have recorded these anomalies from a surgical point of view. Broadly speaking, they may be classified into 3 groups according to the presence of a double aorta, right aorta, or left aorta. Coarctation dominates in the last-mentioned group and may be observed alone or associated with other anomalies, the classic examples being bicuspid valve and cerebral aneurysm (Clagett and Jampolis⁴), as is also persistent ductus Botalli, which according to Abbott⁵ is found in 10 per cent of the cases. This latter finding agrees with that made in materials from recent years (Knutson and associates⁶).

Coarctation mainly occurs in two groups, the adult type with a relatively short stenosis localized below the point of origin of the left subclavian artery, and below or at the level of the ligamentum arteriosum or ductus Botalli. The other main group, which has been termed the infantile or preductile type, is located above the ligamentum arteriosum, and is usually so long that surgical reconstruction is only possible with arterial graft (Gross and co-workers⁷). Frequently, the left common carotid artery and the left subclavian artery spring from the narrow site (Burford⁸). This offers poor or insufficient supply of blood to the left side of the brain, and the preductile type is not usually compatible with life for more than a few months.

We have recently had a case of coarctation of the aorta in which double stenosis was found, with a small intermediate chamber which was the point of origin of the left subclavian artery. Structural conditions were fairly well adapted for success in surgery without many serious difficulties. The immediate result was favorable, and we have therefore considered it justified to record the case here.

CASE RECORD

The patient was a 6 year-old boy, born at full term, who developed normally until he was 5 years old. Then he became unable to take part in play with other children, and he easily became sullen and low spirited ("ungovernable temper," Gerbode and Bourne⁹). He tired easily and his feet were cold. There was no cyanosis.

Clinical Examination—His head was large. There was vigorous pulsation in the right radial artery, and also on the right side of the face. On the left side the pulse was feeble, but countable in the radial artery. There was an invisible, hardly perceptible pulse in the left

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side of the neck and face, no perceptible pulse in the lower extremities, which were cold to the touch, no perceptible or visible collateral vessels on the chest. The blood pressure was 155/90 in the right arm, 100/70 in the left arm, and not measurable in the lower extremities.

A slight fremitus was felt over the heart, and a distinct systolic, blowing sound was heard, loudest in the second and third intercostal spaces at the sternal margin. The sound was heard upward on the right side of the neck, not on the left. There were no special findings by blood examination.

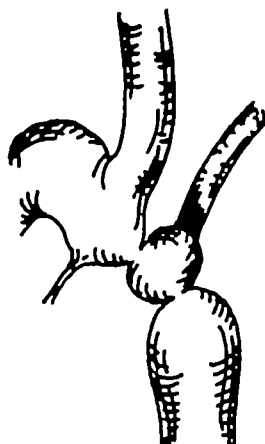
Electrocardiography—Showed right axial deviation, hardly pathological.

Radiography—Demonstrated a slightly enlarged heart, and increased width of the shadow of the vascular trunk with left outline as on a dilated subclavian artery. Strong pulsations in the ascending aorta and an impression in the esophagus at the level of the fifth and sixth ribs were considered to have been caused by a poststenotic dilatation of the descending aorta. Costal impressions were on the right side of the aorta—not on the left.

Because of the clinical and radiographic findings, it was assumed that the stenosis was located above or at the departure of the subclavian artery, and it was considered that the prospects of surgical correction were poor. As we would not deprive him of the chance, however, thoracotomy was performed on June 5, 1953.



A



B

Fig 1—A and B The anatomical findings after the exposure of the aortic arch and the great vessels

The chest wall held strikingly few collaterals. Two vessels arose from the top of the strongly pulsating aortic arch, one of almost the same caliber as the aorta proper (Fig 1). Below the origin of this vessel, another vessel arose, which was of materially smaller caliber. Above and below the departure of this latter vessel, the aorta was greatly constricted, more so below the point of origin. The aorta proper was also narrower than usual at this point.

The pulsations in the largest vessel were very marked, those in the smaller vessel were very feeble. Both vessels had a parallel course, passing up toward the dome. No other vessels had their origin in the aortic arch, and it was thought that the greater vessel was the innominate artery with ample blood supply to the

right arm and the right carotid. The lesser vessel was assumed to be a trunk common to the carotid and subclavian arteries with correspondingly reduced pulsation in the peripheral regions of these vessels.

The duct had its origin above the upper constriction and was of medium dimensions. It was not felt to be patent by palpation. The development of the collateral circulation was strikingly weak with few and small intercostal arteries.

It was decided to perform a side-to-side anastomosis between the innominate and subclavian arteries in order to preserve some circulation while the anastomosis was placed then to ligate the subclavian artery at the point of origin and as the distance between the upper and lower constrictions was fairly short to resect this part of the aorta, and make an end-to-end anastomosis in the usual manner after having first ligated the ductus Botalli. Due to the deficient collateral circulation it was not considered justifiable to ligate the subclavian artery. As previously mentioned, the left carotid artery also originated from this trunk. The operation was performed as planned without essential difficulties (Fig 2).



A



B

Fig 2—A and B The status after surgical reconstruction

There followed immediately a marked improvement in the pulsation in the left arm and the left side of the neck and head. The day after the operation a distinct pulse was felt in both lower extremities which were warmer than previously. The blood pressure in both legs was 140/90, and in both arms 120/80. The postoperative course was uneventful.

Control examination at the end of September 1953 showed normal values of blood pressure in arm and leg equal on both sides. Radiography showed reduction in the size of the heart.

DISCUSSION

Thus in the present case there was the usual departure of the innominate artery. A vessel was present on the left side—a common trunk of

the common carotid and the left subclavian arteries. Above and below the point of departure there was stenosis of the aorta, most marked below (Fig 3). The stenosis above, however, was so extensive that the blood pressure in the left arm was decidedly lower than in the right. Likewise there was no pulsation in the left side of the neck, while it was marked and vigorous in the right side.

Clarke and Dodrill¹⁰ record a case with a common point of origin from the aortic arch of the four arteries (carotid and subclavian to both sides) with simultaneous coarctation. We have not, however, been able to find any case mentioned in the literature with a similar point of origin of a vessel as in the case just presented.



Fig 3—A and B The removed specimen of the aorta showing the stenoses.

Coarctation with unilateral hypertension and normal or low values contralaterally is well known (Weber and Knop,¹¹ Knutson and co workers⁶).

In three cases of coarctation with lacking radial pulsation on the left side, Grishman and associates¹² succeeded in demonstrating by arteriography that there was atresia of the left subclavian artery concurrently. Gerbode and Bourne⁹ gave an account of a similar case, in which the rudimentary subclavian artery was ligated, and the usual anastomosis made, after resection of the stenosed region. Holms and Love¹³ recorded a case associated with stenosis of the right subclavian artery, in which the low values were found on the right side. We have, in our own series of coarctations, a similar case.

Ligation of the common trunk of the carotid and subclavian arteries without anastomosis might possibly have been undertaken, in our case without grave sequelae (Clagett⁴), though gangrene of the arm, after the Blalock operation, has been described (Lam,¹⁴ Webb and Burford¹⁵). However, in due consideration

of the lack of collateral circulation, and of the fact that the carotid artery to the left side also took origin from this branch, we considered it advisable first to place an anastomosis between the innominate and subclavian arteries

SUMMARY

A case of coarctation of the aorta is reported in which there was a double stenosis, above and below the departure of the common artery for the neck and left arm. Resection of the aorta was performed, combined with an anastomosis between the innominate artery and the common trunk of arteries to the left side

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SURGICAL TREATMENT OF CORROSIVE STENOSIS OF THE THORACIC ESOPHAGUS BY SUPRA-AORTIC ESOPHAGOGASTRIC ANASTOMOSIS WITHOUT RESECTION

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A LARGE percentage of corrosive stenoses of the esophagus are in the province of the endoscopist and treatment consists of the following (1) dilatation from above with esophagoscopy control, (2) dilatation along a string, and (3) retrograde dilatation with gastrostomy.

The disadvantages of all endoscopic procedures are the necessity for prolonged treatment, and the ever-present possibilities of mediastinitis from the trauma of dilatation, and of recurrent stenoses. Stenosis may occur and recur many years after the original injury.¹ Frequent dilatations and the chronic irritation from food passing through a corrosive stricture, even without obstruction, have a definite tendency toward the production of carcinoma. Benedict² reported from the literature a series of 236 cases of carcinoma of the esophagus of which 55 per cent followed benign strictures.

The failure of these methods, due to lack of cooperation of the patient, as well as extremely severe or old strictures, demands a surgical reconstructive procedure. Ante-thoracic reconstructions using skin tubes, small intestine, colon, or stomach are multiple stage procedures which require prolonged treatment and the results are uncertain and produce considerable disfigurement.

Intrathoracic reconstruction with resection of the esophagus has been successfully performed with the use of the colon, small intestine, and stomach through the left chest and more recently through the right chest. It occurred to me that leaving the esophagus in situ would cause little difficulty if adequate drainage for the segment were provided, and it was felt that if the segment were not exposed to chronic irritation the danger of carcinoma would be minimized.

Sweet³ very aptly states: "Because of the extreme mediastinitis following lye burns of the esophagus, resection of the esophagus in these patients is unusually difficult and hazardous."

The following hazards are enumerated: (1) tearing of the tortuous esophageal arteries from the aorta, (2) bleeding from the azygous vein, (3) injury to the thoracic duct, (4) entering the opposite side of the chest, (5) production of vago-vagus reflex (this is probably due to a combination of anoxia and other facts but should be considered), (6) late vagus nerve sequelae, and the (7) prolonged operation time.

Moreover, by using a side-to-side technique, it was felt that the anastomosis would be more secure because the esophagus is not mobilized and that it would not tend to retract as with an end-to-end anastomosis. Also, the slitlike opening

produced in a side-to-side anastomosis might tend to prevent regurgitation esophagitis and at the same time could be made sufficiently large to minimize the possibility of stenosis

It was after consideration of these factors that this procedure was decided upon in the following case

A 40 year old white woman was first seen approximately 30 minutes after swallowing a cup of hot black coffee containing approximately 2 tablespoons of commercial lye. She was given vinegar and had profuse emesis. She was hospitalized with severe substernal pain, a white blood count of 20,000, and temperatures to 102° F. Parenteral fluids and antibiotics were used and after 10 days she was able to tolerate a clear liquid diet. She then disappeared until 5 weeks after the injury, when an esophagogram was taken and showed a severe stricture of the esophagus from the aortic arch to the stomach (Figs 1 and 2). She was esophagoscoped and dilatation with soft rubber bougies was begun. She again operated poorly and was not seen until 4 months after the injury when a gastrostomy was performed and retrograde dilatations over a string were begun. She again left town and returned 4 months later (8 months after injury) with the gastrostomy closed and with a weight loss from 114 pounds to 73 pounds. She was again esophagoscoped and the stricture was found to be extremely firm, making surgical reconstruction imperative. She was prepared with blood, protein hydrolysates, and high caloric intravenous fluids. Before operation the protein was 7.2 mg, with albumin 3.62 mg, and globulin 3.50 mg.

Summary of Operative Procedure—The patient was placed in right oblique position. The abdomen was opened, the incision incorporating the previous left gastrostomy incision. The stomach was mobilized by removing the spleen and transecting the gastrocolic ligament and left gastric artery near the celiac axis, care being taken to leave the vessels near the stomach intact as far as possible. The thoracic incision was then made from the angle of the fifth rib following the rib to the costochondral junction and extending inferiorly through the costal cartilages to join the abdominal incision. The lung was then retracted anteriorly and the esophagus very carefully freed above the aortic arch, care being taken to avoid injury to the right pleura or to the thoracic duct. A tube, placed in the esophagus through the mouth, expedited this procedure. The superior intercostal artery of the arch was transected to allow slight depression of the aortic arch. A Penrose drain was then placed around the esophagus in order to draw it into the field of operation. The lower end of the esophagus was clamped and transected and the normal esophageal opening into the stomach was inverted and sutured. The stomach was then brought up to the site for anastomosis which was easy to accomplish with slight mobilization of the left lobe of the liver. There was no question as to the blood supply of the stomach as the proximal end bled freely at the time of anastomosis. A posterior layer of sutures was then placed between the stomach and the upper esophagus and a one and one-half inch incision was made in both organs. The anastomosis was then completed in the usual manner with an outer row of interrupted cotton and an inner row of continuous intestinal catgut sutures. The tube in the esophagus was passed through the site of anastomosis into the stomach.

The distal transected esophagus was then anastomosed to the stomach by placing a small catheter 4 inches in length into the distal esophagus and inserting the esophagus into a stab wound in the stomach near the pylorus. A double row of sutures from the esophagus to the stomach were then placed. The stomach was then attached to the posteromedial thoracic wall with interrupted catgut sutures. The diaphragm was closed snugly about the stomach and the chest and abdominal wall closed in the usual manner. Underwater drainage was established through the ninth intercostal space. One million units of penicillin and 1 Gm. of streptomycin were instilled into the pleural and abdominal cavities before closure.

The patient had an unusually benign postoperative course. She was given Urecholine post-operatively and had no gastric retention, diarrhea, or other vagotomy symptoms, which were anticipated, although no pyloroplasty had been performed. Because of the extensive mobilization, the sympathetic fibers as well as the vagus were interrupted and this probably contributed to the lack of retention. The intrapleural tube was removed on the third postoperative day and the intragastric tube on the sixth postoperative day. She regurgitated the 4 inch

Fig 1

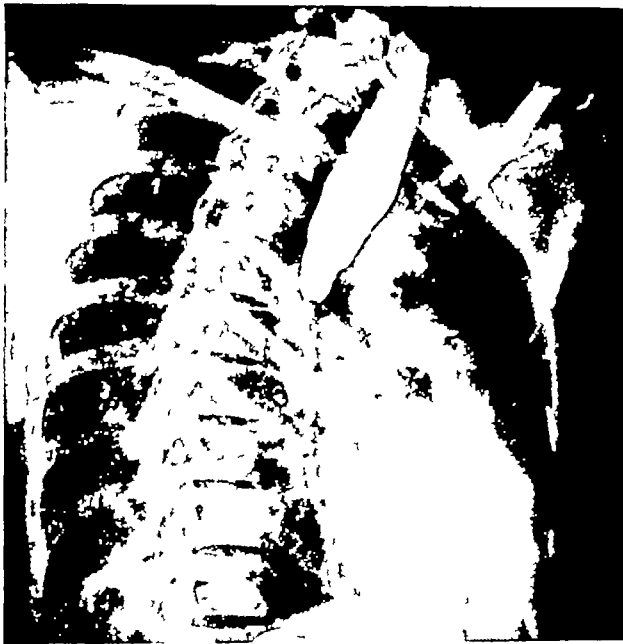


Fig 2

Figs 1 and 2—Oblique and lateral esophagograms showing stricture—numerals on oblique film were taped to skin for purposes of identification

catheter from the lower esophagus several days after being discharged from the hospital. She was able to tolerate a soft diet on the seventh postoperative day and was discharged on the eleventh postoperative day. She rapidly gained weight and denied any regurgitation or other symptoms even when lying on her right side after a meal, with the exception of an occasional slight "soapy" taste (Fig 3).



Fig 3—Chest x-ray taken on the fourth postoperative day. It shows the upper portion of the stomach at the level of the fifth rib posteriorly which has been removed. The catheter in the stomach is the one allowed to remain in the lower esophagogastric anastomosis which has been extruded and is floating free in the stomach. It was later regurgitated through the mouth.

An interesting sidelight upon the case was that this patient was extremely enthusiastic after becoming gainfully employed, and regaining her normal weight, but approximately 14 months after her operation, she was attacked and killed by a gunshot wound in the head. I was able to examine the operative site at post mortem. The upper esophagogastric anastomosis was patent and adequate. The lung was normal with moderate pleural adhesions and adhesions to the stomach. The lower esophagus was completely obliterated so that a probe could not be passed from the stomach to the stenosed esophagus. There was no evidence of a collection of fluid or debris in the esophagus (Figs 4 and 5).

DISCUSSION

Rapant and Hromada⁴ reported 2 cases of esophagogastric anastomosis without resection in which the esophagus was mobilized to the aortic arch, thereby

EXPERIMENTAL TRANSBRONCHIAL CARDIO-ANGIOGRAPHY

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INTRODUCTION

INTRAVENOUS angio-cardiography has been of little help in detecting left-to-right intracardiac shunts,² and it usually does not afford a detailed outline of the left side of the heart and the thoracic aorta. In an effort to improve the results of this method, procedures have been described which consist fundamentally either of injecting the contrast material under pressure into the pulmonary artery—for the visualization of the left atrium and ventricle,⁴ or of taking numerous x-rays in a brief period of time during the injection of an opaque medium—for the diagnosis of interatrial septal defects.⁵ However, in the opinion of its own authors, the former procedure gave no better results than those obtained with Robb and Steinberg's original technique, the latter seems to be a rather complex procedure requiring highly specialized radiological equipment.

It has already been reported⁶ that clear pictures of the left ventricle can be obtained by introducing a contrast substance directly into that chamber. However, the fact that the left atrium cannot be outlined accurately places a definite limitation on this original method.

The purpose of the present study is primarily to extend the applicability of "cardio-angiography."⁶ By injecting a contrast substance into the left atrium this chamber can also be properly visualized, together with the left ventricle and the aorta.

METHOD AND RESULTS

The procedure was carried out in a series of two normal dogs and four dogs with experimentally produced interventricular septal defects. These defects were created by inserting into the base of the interventricular septum a short, rigid plastic tube with a 4 mm diameter bore. The animals were studied from six to twenty-one months after the production of the defect, and physical findings as well as phonocardiographic and cardiac catheterization studies have confirmed the presence of a left-to-right shunt.⁸

After the animals had been anesthetized with pentobarbital sodium and placed in the right posterior oblique position, the left atrium was approached following a technique similar to that described by other investigators^{1,3} for the measurement of left atrial pressures. Accordingly, a Chevalier Jackson bronchoscope was inserted into the trachea until the carina was visualized, and then a 15 gauge needle fitted to a saline-filled aspirating tube was passed through the

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A.



B

Fig 1—4 Chest roentgenogram of a dog with a subaortic interventricular septal defect produced with a plastic tube. The arrow points to the fine wire fixed to the proximal end of the device. The needle is seen in position in the left atrium ready for the injection of the contrast medium.

B Chest roentgenogram of the same dog during cardio-angiography. The tip of the needle is seen in the left atrium. The arrow indicates the contrast material as it passes through the tube from the left to the right ventricle. The left atrium the left ventricle and the aortic arch are well outlined.

bronchoscope and inserted into the left atrium through the wall of the left main stem bronchus at a point 1 cm from the carina. The location of the tip of the needle in the left atrium was confirmed by aspirating bright red blood with a syringe attached to the tube. Then, 70 per cent Diodrast equaling 1 cc per kilogram of body weight was injected rapidly by manual pressure. Films were taken simultaneously with the injection at a rate of four per second, using a Rigler-Watson rapid film changer.*

Electrocardiographic tracings revealed no significant changes either during or following injection of the contrast material.

Films of the normal dogs revealed good detail of the left atrium, left ventricle, arch of the aorta, and brachiocephalic arteries. In the dogs with interventricular septal defects the shunt of contrast material from the left to the right ventricle was detected in every case (Fig 1, A and B).

COMMENT

This preliminary report is submitted because of the success of the procedure in a limited number of dogs, indicating its potential value in proper application to selected human subjects, in the elaboration of abnormalities of the cardiac septum, the left heart chambers, and the aorta. The diagnosis and localization of cardiac septal defects and the differentiation between mitral stenosis and mitral regurgitation may well be facilitated by this method. With the increasing progress of intracardiac surgery, the desirability of more exact methods for diagnosing these lesions has become apparent.

SUMMARY AND CONCLUSION

Cardio-angiography was performed on two normal dogs and four dogs with surgically created interventricular septal defects. The method used was based on the direct injection of contrast solution into the left atrium through the left bronchus under direct visualization through a bronchoscope. Good detail of the left side of the heart, the interventricular septal defects, and the aorta was obtained.

This method may have a valuable potential in the light of recent advances in intracardiac surgery.

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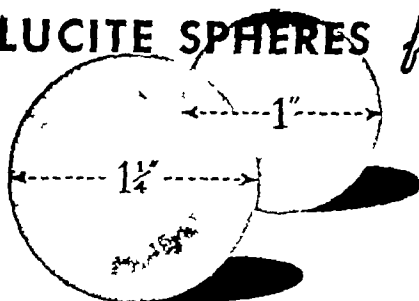
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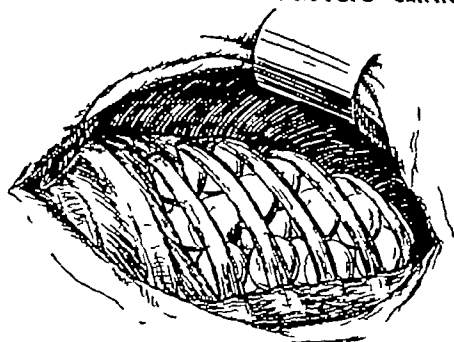
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ANNUAL MEETING NOTES

The Thirty fifth Annual Meeting of the American Association for Thoracic Surgery was held in Atlantic City New Jersey on April 24, 25, and 26 1955. More than 750 physicians registered for the scientific sessions.

The following changes in the By laws were voted at the Executive Session.

A section was added to Article IV of the By laws as follows:

Section 8. Any member of the Association more than 60 years of age is excused from the attendance requirement of Article IV. Section 7 and upon his specific request may likewise be excused from the payment of dues.

Article V. Section 5 was amended so that the final sentence now reads as follows: "The Editor shall be elected from the Honorary Active or Senior members of the Association."

Article VI. Section 2 was amended to read as follows: "The Membership Committee shall consist of seven (instead of six) Active or Senior members and not more than five (instead of four) of the members may be reappointed to succeed themselves."

As a memorial to Dr. Howard Lihenthal, a Founder Member of the Association, a wooden gavel block was presented to the Association by his long time pupil and associate, Dr. Arthur Touroff.

MEMBERSHIP NOTES

At a special meeting of the Council with the entire Membership Committee the following clarification of the standards for election to Associate Membership was made. Copies of these standards which were accepted by the Association at its 1954 meeting were sent to each member of the Association last year.

A. Training

1. Applicants in the United States must have a sound surgical training as evidenced by completion of the American Board of Surgery.
2. Applicants in the United States must further have met the requirements of the Board of Thoracic Surgery. Canadian applicants must have Fellowship in the Royal College of Surgeons, be qualified in thoracic surgery or have passed the Royal College examinations for certification in thoracic surgery.

B. Professional Work

1. High moral and ethical standards are required.
2. Applicant should be established in practice in his community or in a teaching position for *at least three years following training*. His professional work must be of the highest quality.
3. The applicant should have a major interest in thoracic surgery.
4. His professional conduct should be such that he has the support of the members of the Association living in the community.
5. The applicant should show evidence of future participation in meetings by making contributions to the medical literature, advancing the knowledge of thoracic disease and by leadership in furthering thoracic surgery in his community.
6. Certification in thoracic surgery by the Board of Thoracic Surgery or by the Canadian Royal College of Surgeons in itself does not mean fulfillment of the requirements for election to Associate Membership. The other qualifications must be met.

C. Allied Specialists

1. Members of the profession in allied specialties who are outstanding in their respective field may be elected to membership in the Association according to the desirability for such specialists as determined by the Council.

D Foreign Applicants

- 1 Thoracic surgeons residing outside the confines of Canada and the United States and its territories are not eligible for election to the Association except as Honorary Members

E Exception

- 1 Notwithstanding the above qualifications, in exceptional instances the Council may recommend for election a candidate who does not meet all of the above requirements

The following changes in membership status were voted by the Association during its annual Executive Session

In recognition of his outstanding contributions in the field of thoracic surgery and of his long continuous service to the Association, Dr Evarts Ambrose Graham was elected to Honorary Membership

The following Active members were elevated to Senior Status

John B Barnwell	Harlan F Newton
George M Curtis	William F Rienhoff, Jr
Julian A Moore	

The following Associate members were elected to Active membership

Henry K Beecher	Roy G Klepser
David P Boyd	Rutledge Starr Lampson
George V Brindley	Arthur M Olsen
Denton A Cooley	J Karl Poppe
Georges Deshaies	Charles B Ripstein
Howard A Frank	Clifford F Storey
Elliott Harrison	Allan Stranahan
John F Higginson	Wilson Weisel
Alfred Hurwitz	George Willauer
John W Kirklin	

The following candidates were elected to Associate membership

Ralph D Alley, Albany, N Y
 Neil C Andrews, Columbus, Ohio
 H William Clatworthy, Jr, Columbus, Ohio
 George H A Clowes, Jr, Cleveland, Ohio
 Oscar Creech, Jr, Houston, Texas
 Albert C Daniels, San Francisco, Calif
 Franklin H Ellis, Jr, Rochester, Minn
 Joffre Andre Gravel, Quebec City, Que
 Paul F Hausmann, Milwaukee, Wis
 Nathan K Jensen, Minneapolis, Minn
 Thomas N P Johns, Richmond, Va
 Victor H Kunitz, Buffalo, N Y
 John L Keeley, Chicago, Ill
 James E Lewis, Jr, St Louis, Mo
 William P Longmire, Jr, Los Angeles, Calif
 Bertrand W Meyer, Los Angeles, Calif
 Paul Nemir, Jr, Philadelphia, Pa
 Joseph L Robinson, Los Angeles, Calif
 Gordon W Thomas, St Anthony, Newfoundland
 Joseph L Mangiardi, Freeport, N Y
 Melvin M Newman, St Albans, N Y
 Rodman E Taber, Iowa City, Iowa

The following resigned from the Association

Richard Davison

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